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COMMUNICATIONS

CONGENITAL VASCULAR VEILS IN THE VITREOUS

BY

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THE following three cases (six eyes) were discovered by one of us (A. McR.) in the course of routine examination. The patients made no complaint other than the need for a refraction test. In all there was relative amblyopia, 6/12 being the maximum acuity obtainable by correction. The patients' ages were 17, 20 and 21 respectively and there was no history of ocular discomfort or inflammation at any time. All three were males.

The first example (described as Case 3) was seen in 1934. The second (Case 1), seen in 1937, volunteered the information that his brother (Case 2) had also defective sight. He was therefore examined and found to have a similar condition. The similarity of all three cases and the presence of the abnormality in both eyes of two brothers, who also resemble each other strongly in complexion and appearance, place the condition with fair certainty as a developmental one.

In many respects the findings appear to link up with those described in cases of congenital retinal fold on the one hand and with persistent hyaloid artery on the other, and offer evidence in

favour of the theory that congenital retinal fold is due to an abnormality of development of the vitreous.

It seems justifiable to consider these cases as belonging to a distinct group which we have called " congenital vascular veils in the vitreous."

Case 1.—John G., aged 17 years.

Right eye, 6/36 Hm. +3·0 D.
Left eye, 6/12 (2) Hm. +0·75 D. } Vision not improved by glasses.

Right Eye.—There is a thin transparent veil in the inferior temporal quadrant of the eye. This is attached to the retina on either side of the superior temporal vein by two narrow strands of tissue. The vein at this point is very dark and appears to come forward into the veil but cannot be traced further.

The upper border of the veil is fairly sharply defined with a serrated edge due to small projections of tissue. It lies well forward in the vitreous. In the temporal periphery it ends in a mass of mottled paper-white tissue which appears fenestrated. Terminal branches of the superior temporal artery and the inferior temporal vein can be traced into this tissue, which is very similar to that seen in the vitreous near the ora serrata in the cases of congenital retinal fold described by one of us (I. M.) in the *British Journal of Ophthalmology* in December, 1935.

The veil on its nasal side appears to fuse with the retina as if the latter structure had divided into two layers. It is impossible to define any nasal edge to the veil.

Somewhat external to the region in which the veil and retina fuse, branches of the inferior temporal artery and vein come forward into the veil and end close together in a mass of tissue in which fine vessels (?capillaries) can be seen faintly.

The inferior temporal artery is crossed by a branch of the inferior temporal vein before it enters the veil. There is therefore no doubt that in the first part of its course it runs normally in the retina and only leaves this to enter the veil at the line of attachment to this. The transparency of the veil and the fact that the retina can be seen in position through it shows that the veil is not formed of a double fold of retina but is an additional structure lying in front of this. A branch of the inferior temporal vein slightly peripheral to the branch referred to above comes forward into the veil and ends in a brush of small vessels some of which have terminal knobs. Fig. 1 shows the ophthalmoscopic appearance and Fig. 2 a diagram of the probable arrangement of parts in a vertical antero-posterior section to the temporal side of the disc.

The left eye of the same case showed a fragment of a similar filmy transparent veil floating free in the lower temporal quadrant of the vitreous. The fragment is roughly triangular in shape with

FIG. 1.

Ophthalmoscopic appearance of the veil and fundus oculi in the right eye of case 1.

A

B C

FIG. 3.

Case 2. A, B and C are vessels invading the veil. Ophthalmoscopic appearance of veil in right eye of case 2.

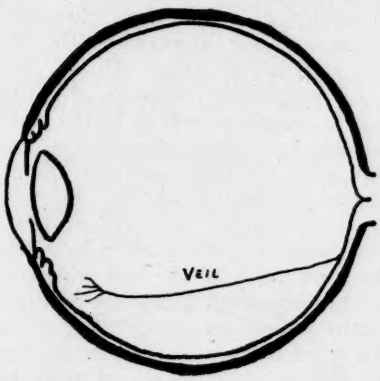

FIG. 2

Diagram of antero-posterior section of right eye of case 1 to temporal side of disc.

the base uppermost. The external and internal angles of the triangle are sharp, the inferior obtuse (120° approx.). No connection with the retina was visible nor could any attachment to the lens be seen. On the film are a few fine lines suggestive of obliterated vessels (probably vasa hyaloidea propria). No other abnormality was seen in this eye but the relatively poor visual acuity (6/12 partly) is noteworthy and shows a definite developmental retardation (retinal amblyopia).

Case 2.—Joseph G., aged 20 years, brother of Case 1.

Right, convergent strabismus.

V.A. Right, C.F.: $\bar{c}$.+2·0 D. sph., +2·0 cyl., axis 180°, no improvement of vision.

V.A. Left, 6/18. Hm. +0·75 D.

Right Eye.—There is a faint white straight streak in the retina starting from the disc about " 9 o'clock " sloping upwards and outwards, and fading out in the periphery. About 2D.D. from the disc a branch of a small supernumerary upper temporal artery curves round towards the disc and lies along this streak. At the point of junction of artery and streak (A in Fig. 3) the veil commences as a very narrow strip of tissue projecting only slightly from the retina till it reaches a point close to the disc. There it leaves the retina and comes steeply forward with the artery visible a little deep to its anterior edge but in places obscured by the tissue. The artery contains blood but cannot be traced beyond this point.

The edge of the film which is now far forward in the vitreous,

curves round from in front of the disc towards the temporal side leaving a small section of the inferior temporal quadrant of the disc clearly visible with no intervening veil tissue. This edge appears dark in the sketch (Fig. 3) but this is an optical effect no doubt due to greater thickness of the film at this point. There is no pigment visible anywhere in the veil.

Just after the edge has turned towards the temporal side there are numerous fine pencilled lines visible in the film running from its edge nasally and slightly upwards. These appear to be the remains of obliterated vessels. The veil narrows from this point into a thin bridge connecting the portion in the neighbourhood of the disc with the main body of the film which occupies the periphery of the inferior temporal quadrant of the eye extending a short distance into the upper temporal and the inferior nasal quadrants. This main portion of the film appears to be attached to the globe in the neighbourhood of the ora serrata, and to stretch across the eye in the vitreous not far from the back of the lens. It is completely transparent, the retina being clearly visible beneath it on focusing down. The somewhat opaque appearance of the film in the sketch is due to an attempt on the part of the artist to achieve the difficult task of conveying an impression of the difference of level between the film and the retina. When the film is sharply in focus with a +9 or +10D. lens in the ophthalmoscope the retina can of course be only dimly seen.

Above the bridge, which runs outwards connecting the part of the veil in front of the disc with the main body of the veil, the edge of the latter is sharply defined with several narrow strands of tissue projecting inwards. The lowest of these is the most substantial and appears dark in the sketch. Below the bridge there is a large hole in the veil. This hole is bordered on the nasal side by a vertical bridge of tissue which runs upwards to join the part of the veil which lies in front of the disc. From this vertical bridge several strands of tissue project inwards. Two of these strands join up with the inferior nasal artery. The lower is obviously a branch of the artery and contains blood. The upper appears to be an obliterated branch of the artery (C and B, Fig. 3).

The mass of tissue in the neighbourhood of the disc has no special features other than those already described. It lies in approximately the same plane as the rest of the veil. Its lower border which forms part of the circumference of the hole is sharply defined. Its upper and inner boundaries are more indefinite. The three points at which vessels leave the retina to enter the veil are indicated by arrows in the sketch (A, B and C).

The peculiar fluffy white tissue in the periphery in John G. is not seen in Joseph G. though far out in the upper temporal quadrant is an area in which the choroid presents a peculiar white

stippled appearance. There is no detachment of the retina which can be seen behind the veil over its whole extent.

Left Eye.—Joseph G. In this eye, as in the left eye of his brother, there is a portion of a veil free in the vitreous. There is some retinal amblyopia (6/18). The veil is floating in the lower half of the vitreous, far forward. It is narrow, never more than 1/3 D.D. in width, but diminishes in places to 1/6 D.D. Starting from a point below and to the nasal side of the disc it runs horizontally to the temporal side and then bends downwards at almost a right angle, ending below in a sharp point. The horizontal

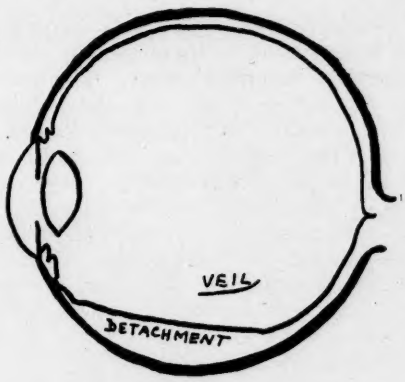

FIG. 4.

Antero-posterior section of left eye of case 2.

and vertical limbs are of approximately the same length. The edges are well defined. There is no attachment to the retina or lens nor are there any obliterated vascular channels in the veil. The retina is everywhere apparently normal except in the inferior part where there is a shallow detachment, extending to the ora serrata, but without a hole. There is a small limitation of the upper field corresponding to this. The detachment has the smooth appearance of a congenital failure of a portion of the inner layer of the optic cup rather than of a secondary displacement. Fig. 4 shows the relationship of the structures in antero-posterior section.

Case 3. George B., aged 21 years. Right eye, 6/24. Left eye, C. F. The right eye shows hypermetropia (+5·0D.) and a condition of hypermetropic pseudo-neuritis.

The left eye (which has +10·5D. hypermetropia) shows a transparent veil similar to that seen in the right eye of the two previous cases. The veil is attached to the retina along a horizontal line

below the optic disc. Both retinal arteries and veins pass forward on to it and it ends far forward in the inferior periphery in a serrated edge which is fluffy and paper-white and similar to the white tissue at the ora serrata in case 1 and in some of the cases of retinal fold. There are patches of choroidal disturbance in other parts of the fundus. Some are white (as in the upper temporal periphery in case 2) and some pigmented like the scars of old choroiditis. The veil in this case is not quite so transparent as in the others and it is not possible to say that the retina is entirely in position behind it. The general similarity with the other cases, however, warrants the supposition that the condition is the same.

Aetiological considerations. The only acquired pathological condition which is suggested by these cases is retinitis proliferans following haemorrhage into the vitreous. The normal appearance of the retina in the region of the veil, the regular arrangement and the absence of a history of progressive loss of vision and the stationary nature of the condition are against this diagnosis. On the other hand, the presence of choroidal changes in the periphery in two eyes and of a shallow detachment in one might be taken as evidence of past, possibly intra-uterine, inflammation. The occurrence of the veil in both eyes of two brothers is definitely in favour of a developmental origin. It can, therefore, be conceded that the condition is a congenital one. We have then to consider the possible embryological meaning of the appearance. The points calling for special examination are:—

1. The nature and origin of the tissue in the veil.
2. The nature and origin of the vessels in the veil.

In the first place the veil may be a purely abnormal structure lying entirely internal to the inner layer of the optic cup and not containing any retinal structures. In favour of this opinion is its extreme translucency, and the fact that in the left eyes of both John and Joseph G. it is floating free on a plane entirely anterior to the retina which can be seen everywhere continuous behind it. Against this opinion however, is the very striking fact that in the right eyes of John and Joseph G. and in the left eye of George B. vessels running in the veil are quite certainly the continuation forward of vessels which are definitely retinal in the first part of their course. The veil therefore, even if not derived from the inner layer of the optic cup, must have been in continuity with this at some time.

It is possible that the retina has split, a thin lamina from its internal surface forming the veil and therefore naturally containing some retinal vessels. The line of splitting, however, presents some difficulties. Any level deep to the internal limiting

membrane would imply a loss of continuity of conducting neurones and would be represented by a corresponding loss of field, which these cases do not show. If the internal limiting membrane alone had split off, this might account for the appearance and we cannot ignore this possibility, though the veil is somewhat too substantial to be composed of this alone. It seems more probable that the veil is of the nature of a vitreous condensation and that, at its commencement, it was in contact with the inner layer of the optic cup. It may be similar to the vascular vitreous condensations sometimes seen in cases of congenital retinal fold. Fig. 5 shows a case (from the *British Journal of Ophthalmology*, December, 1935), of retinal fold in which a thickened portion of the primary vitreous containing vessels was adherent to the retina and had

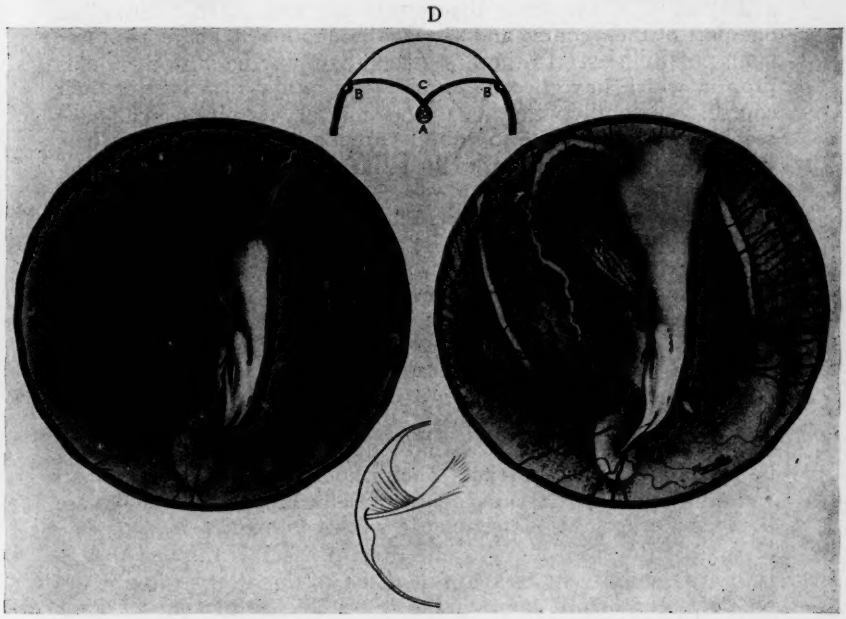

FIG. 5.

Ophthalmoscopic appearance of retinal septum in the left eye of a girl of 9 years. A, as seen with a +12 D. sphere. B, as seen without a lens. C, a diagram showing the vertical disposition of the retinal fold attached to the persistent hyaloid. D, a horizontal section through the fold. (A=the persistent hyaloid in its glial sheath. B=the scar-like adhesion to the pigment epithelium and choroid. C=the tent-like detachment or fold.)

caused a tent-shaped detachment of this over a narrow area above the disc.

The thickened tissue adherent to the apex of the fold in this case ends far forward near the ora serrata in fenestrated white fluffy tissue similar to that seen in cases 1 and 3 of the present series. It therefore seems justifiable to say that the veil is a vitreous derivate showing some affinity with the abnormal condensations of primary vitreous seen in some cases of congenital retinal fold. A similar tissue is also sometimes seen in cases of persistent hyaloid artery. The tissue is not itself retinal but it may play a part in the production of congenital retinal folds and detachments. It is noteworthy that in case 2 there is a peripheral congenital detachment in the lower part of the retina.*

2. The nature and origin of the vessels in the veil. This is a difficult problem, since the vessels are undoubtedly retinal in one part of their course and vitreous in another. There are three main possibilities. In the first place they may be vasa hyaloidea propria which have become secondarily retinal in a part of their course. Secondly, they may be retinal vessels which have secondarily grown into the vitreous condensation where this is abnormally adherent to the retina. Thirdly they may represent an anastomosis between vasa hyaloidea propria and retinal branches of the arteria centralis retinae. In favour of their being vasa hyaloidea propria which have been laid down on the retina in some part of their course is the fact that in many animals (*e.g.*, rats, mice and some snakes), the definitive vascular supply of the retina is derived from the laying down on it of the membrana vasculosa retinae which is composed of the outer set of the vasa hyaloidea propria. It is therefore quite possible that this might occur as an abnormality over a part of the retina in man. If this were to happen it is probable that no further branches of the arteria centralis retinae would grow into this area as it would already be vascularised at the time when this should be happening.† Indeed cases of partial persistence and attachment of isolated vasa hyaloidea propria to the retina are known in man.

Against this possibility in the present cases is the fact that in case 1 a retinal vein crosses superficial to the artery while it lies on the retina before entering the veil. This would not be likely if the vessel were a vitreous one laid down on the retina as then it should be morphologically superficial to the internal limiting membrane while the veins, developed later, would be deep to this.

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* Embryologically speaking, the term "detachment" is wrong. "Failure of coaptation" is more correct.

† The vasa hyaloidea propria are at the height of their development by the 48 mm. stage, the branches of the central artery only appear at 100 mm.

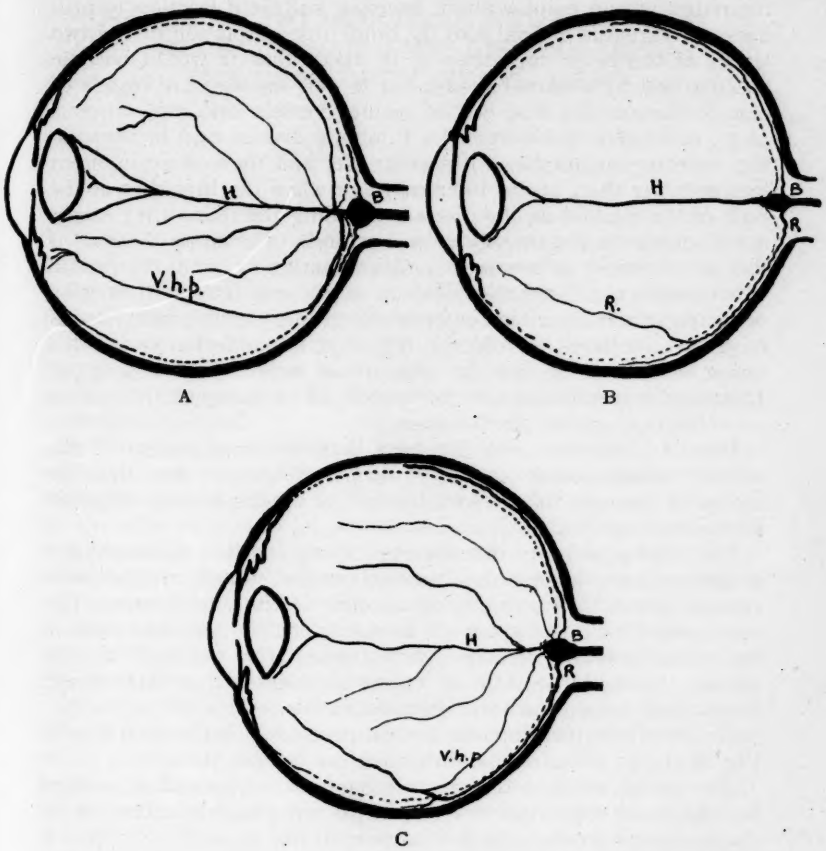

FIG. 6.

Possible modes of production of vascular veils. A. One of the vasa hyaloidea propria in the lower part of the eye becoming retinal in the first part of its course. B. A branch of the arteria centralis retinae running into the vitreous veil. C. An anastomosis between a branch of the central artery and one of the vasa hyaloidea propria. H. Hyaloid. V.h.p. Vasa hyaloidea propria. R. Retinal artery. B. Bulb of hyaloid.

On the whole this explanation does not correspond with the appearances.

There is more to be said in favour of the second possibility that the retinal vessels have grown into the veil secondarily. If a condensation in the primary vitreous had remained adherent to

the retina at one point without exerting sufficient traction to produce a congenital retinal fold by hindering coaptation of the two layers of the optic cup, then it is likely that it would become vascularised by a retinal vessel. It is well known that vessels of new formation can bud out of retinal vessels into the vitreous (*e.g.*, in retinitis proliferans, in Lindau's disease and in organising vitreous haemorrhages) in adult life, and there is no inherent impossibility that, at the 100mm. stage when the branches of the bulb of the hyaloid on the disc are invading the retina they might not also invade abnormally placed strands of primary vitreous if this were closely adherent to and in continuity with the retina. It is possible that since the adhesion might antedate the formation of the membrana limitans interna the proper development of this might be interfered with in the region of the adhesion and so the retina would not be sharply demarcated here. (The absence of Descemet's membrane in the region of a congenital anterior synechia is a similar phenomenon.)

This is consistent with the facts that the arrangement of the retinal vessels seems perfectly normal elsewhere and that the extent of invasion of the veil by retinal vessels is very different in the various eyes.

The third possibility that there is, along the line of attachment of the veil, an anastomosis between retinal vessels and vitreous vessels, is not likely, chiefly on account of the time factor. The vasa hyaloidea propria are all atrophied before the branches of the arteria centralis retinae have extended from the disc into the retina. It would only be in exceptional cases that they would persist long enough to form an anastomosis.

On the whole therefore the second possibility is the most likely. Fig. 6 shows some of the embryological possibilities.

The initial cause and exact mechanism is impossible to state but the cases seem to throw some light on possible anomalies of the primary vitreous and their effects on the retina.

REFERENCE

IDA MANN.– Congenital retinal fold. *Brit. Jl. of Ophthal.*, December, 1935, pp. 641-658.

A CASE OF INTRA-OCULAR NEUROMA (VON RECKLINGHAUSEN'S DISEASE) OF THE LEFT OPTIC NERVE HEAD

BY

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VON RECKLINGHAUSEN'S disease is congenital, slowly progressive and affects males more commonly than females. Ocular complications are evident in some cases of this disease. The characteristic pathological changes are confined to and diffused throughout the distribution of one or more contiguous nerves or a plexus of nerves. The branches of the first division of the fifth cranial nerve, the optic nerve and the nerves to extra-ocular muscles are sometimes affected.

In the orbit the branches of the first division of the fifth become enlarged and convoluted and on palpation of the orbital contents they give the impression of feeling like a bag of worms. The eye on the affected side is proptosed and displaced, often downwards and forwards. Ptosis is evident, the skin of the lids is hypertrophied, the epithelium is thickened, the papillae are enlarged and numerous and there is fibrous tissue hyperplasia in the corium. In some cases the affected area of skin is very hairy and pigmented. The Meibomian glands are widely separated by hypertrophied fibrous tissue, they are irregular in shape and size, contain degenerate vacuolated cells and are surrounded by a few round cells and giant cells which are also present beneath the skin. The blood vessels are numerous, congested and dilated. The circumference of the orbital margin is increased and the orbital walls become thin from pressure atrophy. In severe cases the bony wall is absent posteriorly and the orbital contents are in direct contact with the intracranial structures. This explains the pulsatile exophthalmos, which is not expansile as in an aneurysm in the cavernous sinus; no bruit is audible, and by pressure it may become converted into a pulsatile enophthalmos. In some cases the pulsatile exophthalmos has misled a surgeon into ligaturing the common carotid artery on the affected side. The exophthalmos is worse on excitement. The temporal fossae are full and bulging and on the affected side a doughy sensation is felt on palpation.

The skull is enlarged, brachycephalic, and the foramina through which the diseased nerves pass are grooved or enlarged.

When the ciliary nerves are affected they are enlarged and convoluted before piercing the sclera, but on traversing this structure they resume their normal thickness to revert to their pathological

state on reaching the uveal tract. The choroid is thickened, its stroma is hypertrophied and its vascular character very reduced, the blood vessels being very few and less conspicuous than in the normal choroid. The stroma contains a mass of nucleated fibrous tissue cells and many branched pigment cells. The nerve endings are enlarged, in their centre are convoluted fibres; they are surrounded by a capsule composed of a single layer of flattened nucleated cells and there is a space between this and the surrounding tissues.

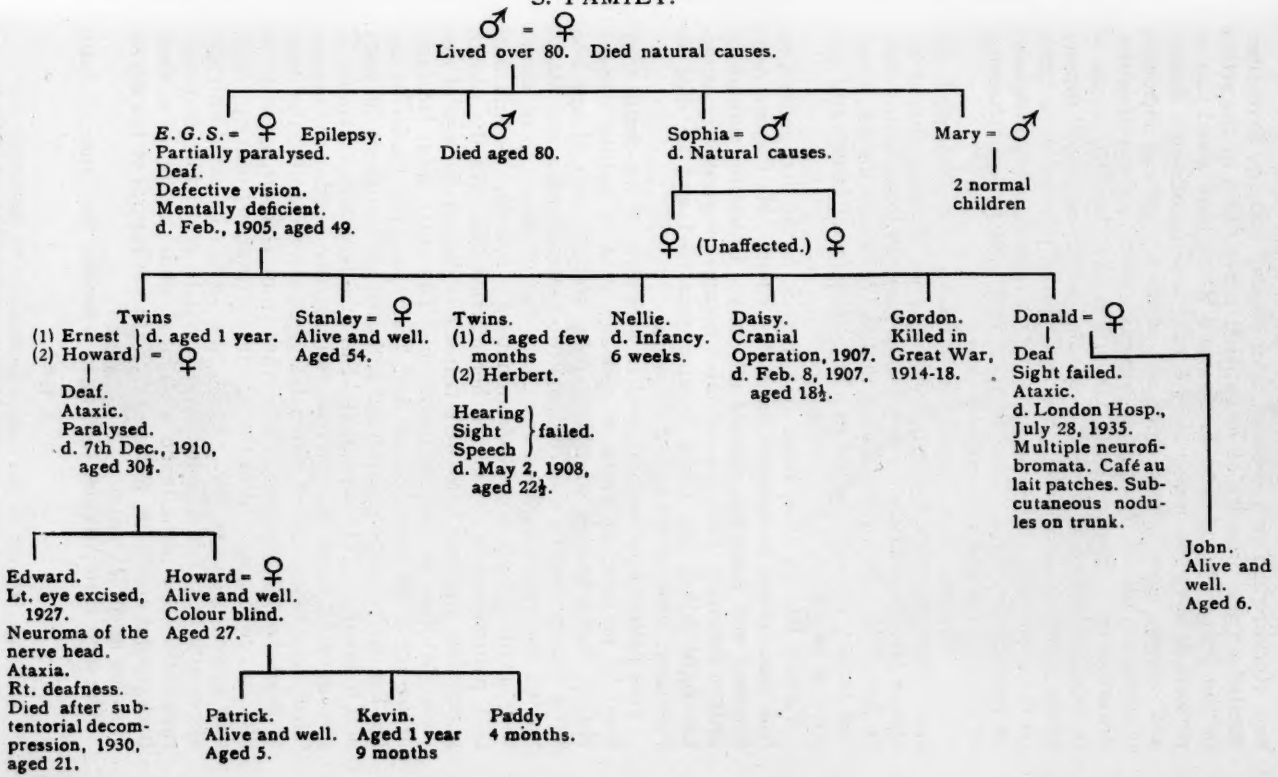
Associated with neurofibromatosis of the ciliary nerves is buphthalmos (infantile glaucoma). An interesting example of the appearance of glaucoma later in life in a subsequent generation affected by von Recklinghausen's disease is given in Achermann's case of a male who at the age of six had the right eye excised for buphthalmos. He had neurofibromatosis of the right upper eyelid and later developed diffuse von Recklinghausen's disease. His son at the age of ten had diffuse von Recklinghausen's disease with no involvement of the lids and at the age of thirty-four years chronic glaucoma developed in the left eye and when he was forty-four years old the right eye became glaucomatous. The latter's son (grandson of the original patient) showed no evidence of von Recklinghausen's disease or glaucoma.

Neurofibromatosis may also affect the nerves of the iris and with slit-lamp microscopy small nodules have been noted on some of the corneal nerves. Similar changes have also been observed in the nerves traversing the outer layers of the sclera.

When the optic nerve is affected by neurofibromatosis both the orbital and intracranial part may be involved causing proptosis and enlargement of the optic foramen and canal. The growth may be pear-shaped with its base towards the globe, or fusiform. Fibrous tissue and glial proliferation, areas of myxomatous degeneration and small cysts are some of the pathological features of neurofibromatosis of the optic nerve. Small greyish-white nodules composed of neurofibrils, neurocytes, glial tissue, cysts and in some cases areas of degeneration and angiomatous elements, may be present on the edge of the optic disc and in the retina but these are rare. The case described below concerns such a lesion affecting the optic nerve head in one eye in a young male, aged 19, who had a marked family history of von Recklinghausen's disease affecting two generations of his forebears and who subsequently died three years later from a subtentorial growth which affected his hearing and gait.

It is well known that café au lait patches in the skin, bronzing, shagreen areas, freckles, moles and naevi are seen in association with von Recklinghausen's disease. Goldstein and Wexler have noted melanoma of the iris and melanosis uveae in connection with

S. FAMILY.


♂ = ♀
Lived over 80. Died natural causes.
E. G. S. = ♀ Epilepsy. Partially paralysed. Deaf. Defective vision. Mentally deficient. d. Feb., 1905, aged 49.
♂ Died aged 80.
Sophia = ♂ d. Natural causes.
Mary = ♂
2 normal children
♀ (Unaffected.) ♀
Twins
(1) Ernest
(2) Howard = ♀
Deaf. Ataxic. Paralysed. d. 7th Dec., 1910, aged 30½.
d. aged 1 year.
Stanley = ♀ Alive and well. Aged 54.
Twins.
(1) d. aged few months
(2) Herbert.
Hearing Sight Speech failed.
d. May 2, 1908, aged 22½.
Nellie. d. Infancy. 6 weeks.
Daisy. Cranial Operation, 1907. d. Feb. 8, 1907, aged 18½.
Gordon. Killed in Great War, 1914-18.
Donald = ♀
Deaf
Sight failed.
Ataxic.
d. London Hosp., July 28, 1935.
Multiple neurofibromata. Café au lait patches. Subcutaneous nodules on trunk.
John. Alive and well. Aged 6.
Edward. Lt. eye excised, 1927. Neuroma of the nerve head. Ataxia. Rt. deafness. Died after subtentorial decompression, 1930, aged 21.
Howard = ♀ Alive and well. Colour blind. Aged 27.
Patrick. Alive and well. Aged 5.
Kevin. Aged 1 year 9 months
Paddy 4 months.

von Recklinghausen's disease, and several authors have commented on the presence of medullated nerve fibres in the retina in this disease. Copeland, Graves and Reese have noted that in persons showing the association of von Recklinghausen's disease and opaque nerve fibres there is a relatively greater incidence of abnormalities of the central nervous system and psychopathic disturbances. Shapland and Greenfield have reported a case of intracranial neurofibromatosis associated with a meningeal tumour of the middle cranial fossa which had spread into the optic nerve on the same side. In some cases sarcoma becomes superimposed on neurofibromatosis. Ocular palsies may arise from involvement of the 3rd, 4th and 6th cranial nerves.

Case Report.—E.S., aged 19 years, a clerk, attended the ophthalmic department at St. Bartholomew's Hospital, under the care of Mr. Rupert Scott (whose House-surgeon I was at this time) on June 17th, 1927, complaining that for 2-3 weeks the left vision had been " smudgy." He was in good general health and had not lost weight.

Family History (See Table A).—E.G.S. in the first generation had been subject to epileptic fits in his youth. He became deaf, squinted and later was affected mentally. From the autumn of 1904 to February, 1905, his condition became progressively worse, his sight failed, his limbs became paralysed and he died in February, 1905, at the age of 49 years.

This patient's father and mother were alive at his death, they lived to be over 80 years of age and died of natural causes. E.G.S. had a brother who lived to be over 80 years of age and left a " large healthy family," and two sisters, Sophia and Mary. Sophia had two daughters and Mary two children, all of whom were unaffected by von Recklinghausen's disease. Sophia died of " natural causes " early in her married life. In the second generation, the first born were twins, one died at the age of one year, the cause of death is unknown. The other twin, Howard, at the age of 29 years became deaf, ataxic and was unable to use his hands, he became paralysed and died at the age of 30 years and 6 months. The second child in this generation, Stanley, is now aged 54 years, is alive and well, married but has no family. Then came twins, one of whom died aged a few months, the cause of death being unknown. The other twin, Herbert, grew to be 6ft. 2in. in height and at the age of fourteen his hearing, sight and speech began to fail. At 20 years of age he could only get about with help, his speech could scarcely be understood and communication had to be made with large white letters on a black background held close to his eyes. He lost control of his saliva. He died aged 22½ years.

The next child, Nellie, died aged 6 weeks, the cause of death

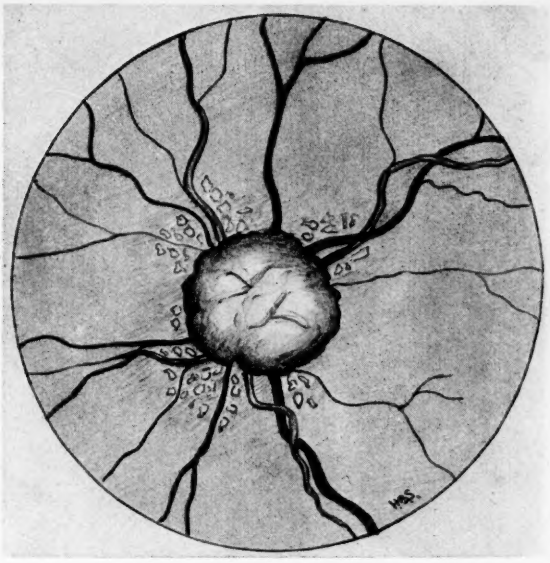


FIG. 1.

Drawing of left optic disc and adjacent fundus.

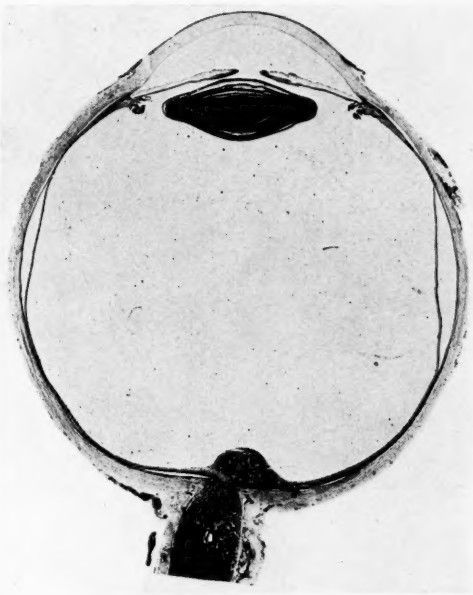


FIG. 2.

Microphotograph of a horizontal section through the left eye. The neoplasm arising from the optic nerve head is shown.

FIG. 3.

Microphotograph (X72) to show the structure of the neoplasm at its summit. Note the cystic spaces, clefts, bunches of neurocytes and neurofibrils and areas of hyaline degeneration.

spaces, clefts, bunches of neurocytes and neurofibrils and areas of hyaline degeneration.

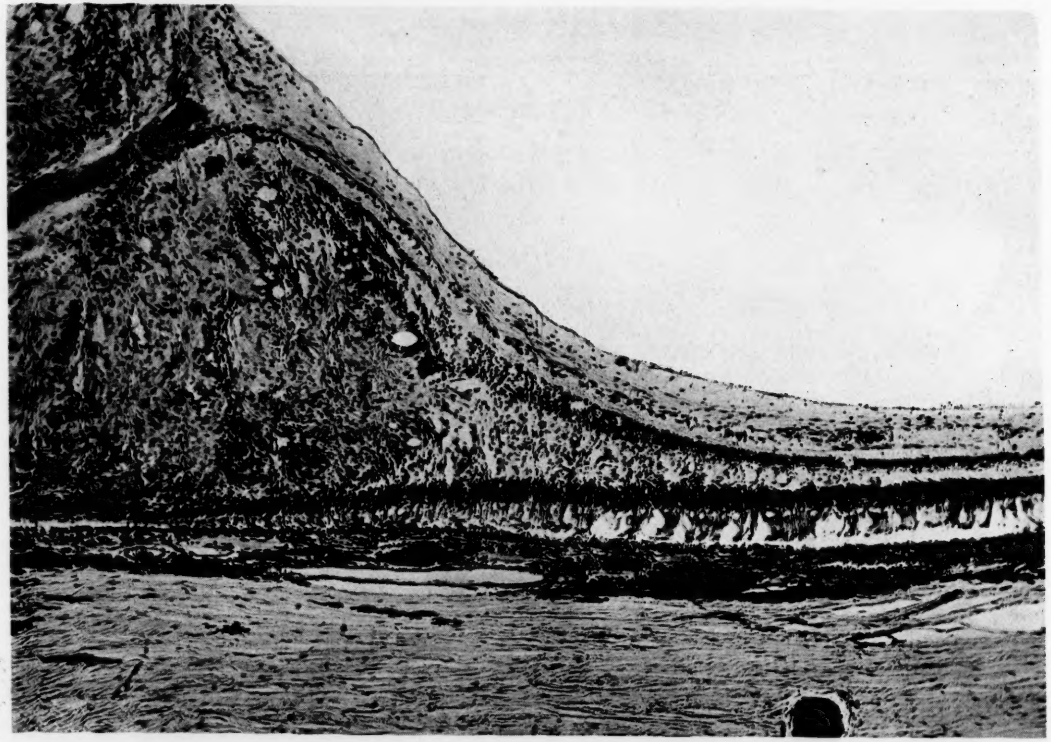


FIG. 4.

Microphotograph (×72) to show the temporal edge of the neoplasm and the changes in the retina and choroid.

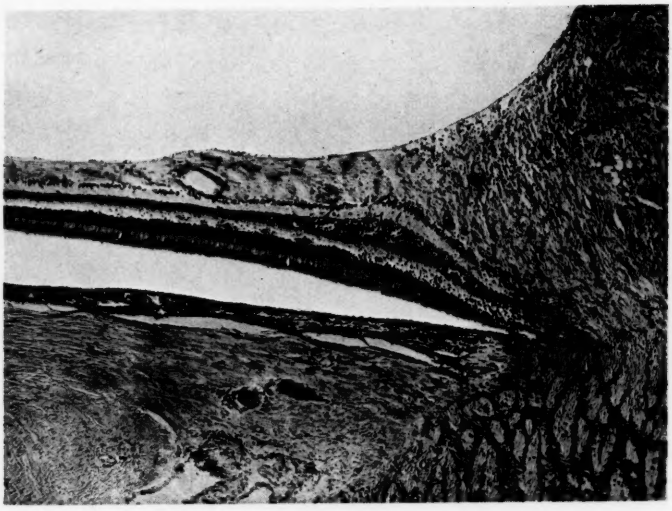


FIG. 5.

Microphotograph of the nasal edge of the neoplasm showing a cluster of small cysts

FIG. 6.

Microphotograph showing the base of the neoplasm. On the temporal side, the retina is extensively destroyed and the choroid compressed. The lamina cribrosa and optic nerve have not been infiltrated by the neoplasm.

being unknown. Then came Daisy, who was well until she was 18 years of age when her fingers became stiff, and she was languid, nervous, somnolent and without energy. She died at the age of 18½ years after an intracranial operation. I have been unable to trace any clinical data about her at the hospital she attended. Gordon was killed in the European War, 1914-1918. Donald, the last child of this generation, at the age of 37 became deaf, ataxic and his sight failed. He died in the London Hospital aged 38½ years after an intracranial operation. His son John is now aged 6 and is alive and well.

Also in the third generation is E.S., the patient whose case is described in this paper. His brother Howard is aged 27, colour-blind, alive and well and married. He has three children, Patrick aged 5, Kevin aged 1 year 9 months, and Paddy a girl aged 4 months. These children are well and up to the present show no evidence of neurofibromatosis.

Condition on Examination.—The left eye of E.S. was slightly divergent and there was also a slight degree of ptosis. The left vision was 6/18 with correction. Fig. 1 is a drawing of the left optic disc and its adjacent structures. Projecting forwards from the optic disc was a hemispherical mass of newly formed tissue, greyish in colour with a nodular surface, and ill-defined outline and edges. Some small irregular flecks of exudate were present in the retina adjacent to the optic disc, particularly on the nasal side and near the main branches of the central retinal vessels. The retinal veins were dilated and engorged with blood. The peripheral visual field was full but there was a paracentral scotoma on the temporal side involving the blind spot and extending towards the fixation point and also downwards. The right eye was normal, the right vision being 6/6. A general medical examination revealed no abnormality. A malignant neoplasm of the left optic nerve head was suspected and the left eye excised by Mr. Scott on June 30th, 1927.

Pathological Report.—Fig. 2 shows a hemispherical neoplasm arising from the optic nerve head and infiltrating the retina on the temporal side. Its diameter is 4·5 mm. and it is 1·5 mm. from its base to the highest point on the summit. The neoplasm has a greyish colour and its consistency on cutting resembles that of the optic nerve.

Fig. 3 is a section through the growth at its summit. It is composed of irregular-shaped clumps and sweeping bundles of neurocytes and neurofibrils. Many small cysts are evident, some being roughly circular and others oval, irregular and cleft-like. These are disposed singly, some approximate each other, and at one site there is a cluster of seven small round cysts. The branches of the central retinal vessels traverse the neoplasm and apart from some

congestion in the larger veins they show no pathological features. Areas of hyaline degeneration are present.

Fig. 4 shows the infiltration of the retina to the temporal side of the optic disc. The growth has invaded all the layers of the retina and the advancing edge has wedged itself laterally between the nuclear layers. At its base the layer of rods and cones has been destroyed, and the retinal pigment epithelium and the choroid are compressed. Fig. 4 shows degenerative changes in the rods and cones beyond the temporal limit of the neoplasm. Over this area many cone nuclei have been extruded through the external limiting membrane into the layer of rods and cones. On the temporal side in the molecular layer there are some small irregular masses of exudate and many swollen cells, arranged in clumps having a faintly granular cytoplasm and an eccentric nucleus. Exudates are also present in the outer molecular layer for a distance of 1 mm. from the optic disc on the nasal side.

The lamina cribrosa is normal in structure and position and is not infiltrated by the neoplasm. The choroid beneath the growth on the temporal side has some condensation of its stroma and areas of hyaline degeneration. Figs. 5 and 6 are microphotographs of the nasal edge of the neoplasm and of its base.

Pathological Diagnosis.—Neuroma of the optic nerve head.

The patient's progress was uneventful until October, 1929, when he first noticed unsteadiness of gait, impaired hearing on the right side, nervousness and attacks of blurred vision in his remaining eye. The impaired hearing became progressively worse and about the middle of May, 1930, he began to suffer from frequent attacks of temporary blindness lasting for half a minute and generalised headaches on rising in the morning. Since June 1st, 1930, he had shown mental disturbance making absurd statements and laughing unreasonably at times. His gait had become unsteady.

The right eye showed coarse nystagmus in all directions and there was well marked papilloedema of the type associated with raised intracranial pressure, but there were no haemorrhages or exudates. The right vision was 6/9.

The late Mr. Theodore Just made an aural examination and noted right middle ear deafness and was inclined to locate the intracranial lesion in the left posterior fossa. An examination of the remainder of the central nervous system, Wassermann reaction and Sigma reaction did not reveal any abnormality. On June 14th, 1930, he became more ataxic, walking on a wide base, was unable to stand on one leg but had no definite tendency to fall to one side.

On June 16th he had a severe generalised headache and one attack of projectile vomiting. The blood pressure rose from 145/90 on June 3rd to 170 systolic on June 20th.

On June 21st Mr. Paterson Ross performed sub-occipital exploration but had to abandon the operation after a bilateral sub-tentorial decompression and tapping the left lateral ventricle, on account of low blood pressure and the condition of the patient. Cerebro-spinal fluid came through the trocar inserted in the left ventricle under considerable pressure and it was noted that the cortex was very thin. After operation the temperature rose to 104°·2, the pulse to 140 and respiration rate 25. On the following day the temperature fell from 103°·4 to 101°·2 in the afternoon but rose again in the evening to 102°·8; the pulse ascended to 160 on the evening of this day and the respiration rate to 40. He died early on the following morning, the second day after operation and unfortunately the relations refused to allow a post-mortem examination.

Commentary.—I have been unable to obtain histological verification about the precise nature of the multiple neoplasms which affected E.G.S. in the first generation and those affected in the second generation with the exception of Donald who died after an intra-cranial operation in the London Hospital and was found at post-mortem examination to have multiple neurofibromata, a few pigmented hairy papillomata in the skin of the ventral wall of the chest and abdomen, some café au lait patches, a faint generalised brownish pigmentation of the skin and a few small subcutaneous nodules on the trunk. Also I have been unable to trace authentic medical information about the cause of death in two infants in the second generation.

In this family there seems to have been a predilection for the multiple neoplasms to affect the central nervous system and particularly the nerves serving the special senses of sight and hearing. As the main clinical features of the disease affecting certain members of these three generations have considerable similarity, it is I think justifiable to assume that the pathological lesion was of the same character in all of them.

It is regrettable that details of complete post-mortem examinations in all these cases have not been available. It is interesting to note that 4 out of the 6 who were affected died between the ages of 18½ and 30½, the other 2 at 49 and 38 years of age respectively. The incidence of visual failure, deafness, ataxia, paralysis, epilepsy and mental symptoms is to be seen at a glance in Table A.

Intra-ocular lesions in von Recklinghausen's disease are very rare. They are briefly mentioned in the literature but I have not found any statistical record of their incidence in this disease.

Summary

A case of neuroma affecting the optic nerve head of the left eye in a young man, aged 19 years, is reported. His family history showed that in the two generations preceding his there were cases of multiple neurofibromata affecting the central nervous system and the nerves serving the special senses of sight and hearing. The histo-pathology of the neoplasm affecting the optic nerve head is described and microphotographs illustrating these changes are shown. Three years after the disease had been discovered in his left eye he developed signs of raised intracranial pressure from a neoplasm or possibly several neoplasms inside the skull. The right auditory nerve was involved. A subtentorial decompression was followed two days later by death.

I thank Mr. Rupert Scott for his permission to publish this case.

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THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY. PRELIMINARY SURVEY OF HEREDITARY EYE DISEASES IN TASMANIA*

BY

J. BRUCE HAMILTON

HOBART, TASMANIA

Geographical

The island of Tasmania is heart-shaped in outline, and has an area of 26,215 square miles and a population of 227,599. The capital—Hobart—which is in the south of the island, has a population of 60,406 and a surrounding urban population of about 50,000. In the north of the island are roughly another 110,000 people—32,841 of whom live in the city of Launceston, the chief civic centre of that part of the island.

Historical

Although Van Diemen's Land was discovered by a Dutchman —Abel Jansen Tasman—on November 24, 1642, yet it was the English who formed in 1803 the first settlement on the island, which has remained a British possession ever since. At first it was entirely a penal settlement, but by 1815 free settlers had commenced to arrive, and in 1853 transportation of convicts terminated. In 1856 the island was declared a Crown Colony, and the name changed to Tasmania in memory of its discoverer; while in 1901 a Federation of Australian States and Tasmania was formed, and this Federation is now known as the Commonwealth of Australia.

It appears to be a significant fact that while Colonel David Collins was founding the first permanent settlement in Tasmania, in the form of a penal settlement in July, 1804, John Cunningham Saunders was circulating to the wealthy merchants of London his appealing letter dated October 1, 1804, which resulted in the foundation of Moorfields Eye Hospital in March of the following year. In St. David's Park, Hobart, stand several historic monuments, on one of which is inscribed the following epitaph:—

" To the memory of David Collins, Esq., Lieutenant Governor of the Colony, and Lieutenant Colonel of the Royal Marine Forces. On the first establishment of the Colony of New South Wales he was employed as Deputy Judge Advocate. And in the year 1803 he was entrusted by His Majesty's Government with the command of an expedition destined to form a settlement at

* The Gifford Edmunds Prize, 1936.

Port Phillip on the south coast of New Holland, which was subsequently removed to Van Diemen's Land. Under his direction as Lieutenant Governor the site of this town was chosen, and the foundation of its first building laid in 1804. He died here on March 28, 1810, aged 56 years, and this monument long projected, was erected to his memory in 1838, by direction of His Excellency Sir John Franklin, K.C.H.K.R. Site of the first church erected in 1810 in Van Diemen's Land. Built over the grave of Lieutenant Governor Collins, whose body rested beneath the Altar."

In the present hall of the Royal London Ophthalmic (Moorfields) Hospital stands a bust with the curt inscription, " John Cunningham Saunders—Founder, MDCCCIV. This bust erected —March 25th, 1845."

But unfortunately the progress of Tasmania was very retarded, for it was not until 1853 that the transportation of convicts to the island was abolished, and the island commenced its history as a Colony of the Crown. The progress of ophthalmology in the island was equally retarded, and it was not until the advent of the present decade that there was a surgeon in the island who devoted his entire attention to eye work. Unfortunately no serious attempt has been made up to the present to record local observations on eye disease, and I am able to find in the literature only one article on Hereditary Eye Disease in Tasmania.

Consequently this survey has been a pioneer work, and an extremely difficult one; first because naturally many of the local inhabitants are very reticent about their antecedents, and resent strongly any probings into the past; and secondly, because the population is so sparse and scattered. (It is well to remember here that the total population of Tasmania is only 1/30 of that of London); and thirdly, relatives of diseased persons reside in many parts of Australia which are beyond the reach of an ophthalmic surgeon. As a result, most of my pedigrees to date are short, except for one or two obvious exceptions, and these exceptions have taken much tact and time to obtain.

One has only to read Usher's Bowman Lecture of 1935, to realise with what infinite pains Nettleship and his disciples have striven in an attempt to correlate their findings, and to present the subject of heredity from the ocular standpoint. When I first read Julia Bell's monograph, I felt that the subject had been more or less exhaustively dealt with. Now that I have perused the literature to some extent I find that so far as she went, Julia Bell acquitted herself well, but unfortunately her work was not completed. There is no monograph in the Nettleship Memorial volume on congenital cataracts, ptosis, epicanthus, or squint, or coming down to the bread and butter of every oculist—on refractive errors. This gap should be filled in as speedily as possible.

Statistical

According to the Commonwealth Census of 1933, the population of Tasmania was estimated as 227,599, and the Commonwealth Bureau of Census and Statistics has supplied me with the following vital statistics in regard to Tasmania:—

Number of people in Tasmania		227,599
Number of people born in Australia (only available)		214,839
Number born outside Australia, excluding 389 birth-place unspecified		12,371
Nationality of place:—British		227,258
	Chinese	104
	Italian	80
	German	23
	United States of America...	26
	Others	108

Except for a few Chinese gardeners and one or two half-castes from the Strait Islands of Flinders and Cape Barren, the population of Tasmania is purely Caucasian, and almost purely British stock; so that the pedigrees to follow should and do closely correspond to those more elaborate ones worked out by Nettleship in England; and Usher in Scotland.

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Heredity and Ophthalmology

The proportion of hereditary disease in Tasmania is difficult to define, but I shall presently attempt to make a rough estimate. On the other hand the preponderance of hereditary blindness in this State, which will be discussed later, can be defined with much greater certainty.

Of 4,880 private patients with eye diseases, seen by me during the last 5½ years—119 have proved to be authentically hereditary in origin, which makes a case incidence of 2·44 per cent., or roughly 2½ in every hundred. This figure I am sure is somewhat low, chiefly due to the fact that during the first 3½ years I was not vitally interested in the subject of heredity, and therefore did not take much pains to elicit family histories from patients who

did not volunteer the information or in whose interest the matter of heredity was of little or no clinical importance. Of course 119 is not the total number of patients examined for these pedigrees, for many others have been examined by me in hospital work, or by my colleagues or their predecessors in private practice in Tasmania.

Diseases associated with Hereditary Eye Defects

The association of hereditary defects of the eye, with hereditary defects in other parts of the body is a peculiarly interesting branch of the subject under discussion, and yet it appears to have received

TABLE I

Hereditary Abnormalities of the Nervous System, associated with Eye Defects

NERVOUS SYSTEM	EYE
Amaurotic family idiocy	Cherry red spot at macula and optic atrophy
Angiomatous cysts of the cerebellum. (Lindau's disease)	Angiomatosis of the retina
Cerebral angioma	Buphthalmos
Dystrophia myotonica	Cataract
Familial hypertrophic neuritis (Dejerine-Sottas)	Nystagmus and abnormal pupil reactions
Friedreich's ataxia	Nystagmus
Hepato-lenticular degeneration (Wilson' disease)	Kayser-Fleischer ring of cornea
Hereditary cerebellar ataxia	Optic atrophy
Mental retardation	Lens opacities Colour blindness High astigmatism Myopia Squint Albinism Opaque nerve fibres
Mongolism	Keratoconus
Neurofibromatosis. (von Recklinghausen's disease)	Tumours of retina and iris
Tuberose sclerosis. (Bourneville's disease)	Cysts of the retina

scant appreciation. Accordingly I append three tables of such associated defects met in the literature, and possibly other writers could add further examples of their own. More particularly does hereditary eye disease appear to be associated with corresponding defects of the nervous and skeletal systems (see Tables I and II), but outside these categories there are a few other associated diseases, especially the Laurence-Moon-Biedl-Syndrome. (See Table III. These will be referred to again.

TABLE II

Hereditary Digital Anomalies in relation to Eye Defects

DIGITS	EYE
Syndactyly	Aniridia
Arachnodactyly	Ectopia lentis
Polydactyly	Retinitis pigmentosa
Brachydactyly	Microcornea
Doubling of thumbs with atrophy of terminal phalanges	Bilateral macular coloboma

TABLE III

Miscellaneous Hereditary Abnormalities, associated with Eye Defects

MISCELLANEOUS	EYE
Aortic stenosis	Ectopia lentis
Eighth nerve deafness	Retinitis pigmentosa
Fragilitas ossium	Blue sclerotics
Harelip	Myopia Microphthalmos
Hypogenitalism and obesity (Laurence-Moon-Biedl syndrome)	Retinitis pigmentosa
Otosclerosis	Blue sclerotics
Stammering	Nystagmus

Possible Hereditary Eye Defects

From Ruggles Gates (1929), and Duke-Elder (1934), and Van Duyse (1933) I have been been able to tabulate the following 44 hereditary eye diseases in alphabetical order.

O Albinism.
Amaurotic family idiocy.
Angiomatosis of retina (Lindau's Disease).
O Aniridia.
Anophthalmos.
Arcus juvenilis (Embryotoxon).
X Astigmatism.
Blue sclerotics.
O Buphthalmos.
X Cataracts, congenital (including Dystrophia Myotonica).
Cataracts—senile.
Choroideremia.
O Coloboma of iris, lens and choroid.
O Colour blindness.
Corneal opacities—nodular.
Corneal pigmentation (hepato-lenticular degeneration).
Cryptophthalmos.
Distichiasis.
O Ectopia lentis.
O Epicanthus.
Glaucoma. Congenital (not buphthalmos).
X Glaucoma. Senile.
Glioma of retina.
Gyrate atrophy of retina and choroid.
O Hypermetropia.
X Keratoconus.
Lagophthalmos.
O Megalocornea.
Microcornea.
X Microphthalmos.
X Myopia.
Night blindness.—Stationary.
X Nystagmus.
Optic atrophy.—Congenital.
X Optic atrophy. Hereditary (Leber's Disease).
Ophthalmoplegia.
X Pterygium.
X Ptosis.
X Retinal detachment.

X Retinitis pigmentosa.
Retinitis pigmentosa sine pigmento.
Retinitis punctata albescens.
X Strabismus.
Tapeto-retinal degeneration (Weber).

In this list I have marked (X) against those diseases of which I have been able to obtain pedigrees in Tasmania; while (O) indicates that isolated cases of these diseases have been examined by me, but no definite hereditary evidence obtained.

Before dealing with each of these thirteen hereditary eye diseases, I propose to examine the causes of hereditary blindness in Tasmania, and the sociological problems of those afflicted in this State. When this is accomplished, then the diseases will be discussed in conjunction with their respective pedigrees.

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Causes of Hereditary Blindness in Tasmania

While ophthalmia neonatorum, trachoma, and smallpox are the three main causes of blindness in Europe, Asia and Africa, these causes are insignificant in Tasmania. First ophthalmia neonatorum of the true gonococcal type is rare in this island, and only

one child between 7 and 16 is at present suffering from blindness as the result of this disease; and actually the exact causal organism of his ophthalmia is not known with certainty. Secondly, trachoma is almost non-existent, and I have only seen four fresh cases and four old ones in 5½ years of private practice, and none in public hospital work. Thirdly, smallpox does not exist in Tasmania at all.

If we review the blind in Tasmania, we will find that blindness in the island is attributable to a considerable extent to hereditary defects.

However disabling most hereditary eye diseases may be, those which cause blindness are paramount in importance, for it is common knowledge that guided by our special senses, our existence depends say 4/5 on eye sight, and 1/5 on the other senses. I have therefore no doubt in writing that the blind are at least 80 per cent. handicapped, and when Best (1934) assures us that only 7 per cent. of the blind in the United States of America are self supporting, and the League of Nations Reports (1929) that 65 per cent. of the blind are unemployable, I feel this figure of 80 per cent. may be too low. To my mind the greatest significance of heredity in ophthalmology lies in the question—does or does not each hereditary disease cause blindness?

The definition of blindness throughout the world varies greatly, but I will refer briefly here to the definition employed by the Government of the Commonwealth of Australia. It is, " Loss or diminution of vision to an extent which renders the sufferer unfit for work other than that usually performed by blind workers." With this definition as a guiding principle, certification for blind pensions is made throughout Australia, and the survey made by W. D. Counsell and myself (1936), into the causation of blindness in Tasmania, was carried out on the principle of this definition. A second difficulty in estimating the causation of blindness, is lack of an international classification of causes. We found when making our review that one disease might be put under several headings. A third difficulty was the incomplete data which was due to two factors: (*a*) certification by incompetent referees, (*b*) vague and incomplete certification forms.

A fourth difficulty was that many of the aged blind received old age pensions, and not blind pensions. And fifthly, the personal factor, *i.e.*, that competent referees vary in their opinion of causation.

The survey made for the Commonwealth Health Council by W. D. Counsell and myself (1936) into the causes of blindness in Tasmania (now published in the 1st Report of the National Health and Medical Research Council of Australia) reveals at least 170 cases from pension and other sources. (See Table IV.) Of these

170, no less than 78 were unreliable in clinical data, and of these 78 no mention was made at all of the causation in 36 cases. Of the remaining 92, no less than 35 were blind from hereditary causes which is in the surprisingly high proportion of 38·04 per cent. Of these 35 cases, 19 were blind from hereditary cataracts, 10 from Leber's disease, 3 from retinitis pigmentosa, and 2 from detachments of the retina, and one from nystagmus. (See Table V.) I could easily have raised this percentage by including glaucoma and myopia, but 37·7 per cent. appears to me to be quite high enough to warrant action. The necessary action will be discussed under prevention, but it suffices to say here that a further 23 of these 92 cases appear to me to be preventable, thus making a total of 58 or 63·04 per cent. of definitely preventable cases. I consider it the privilege and duty of the medical profession to act, and to act quickly in this matter. The public are now sufficiently enlightened to co-operate if carefully led.

In conclusion let me quote Best (1934), in his summary of the prevention of blindness in general and not hereditary blindness in particular:—

"Thus we find what seems to be a fair estimate, 72 per cent. or not far short of three-fourths of the blindness in the United States of America to be of preventable character."

My minimal estimate for Tasmania is 63·04 per cent. (see Table IV), which is just two-thirds.

TABLE IV

Major Causes of Blindness in Tasmania

CAUSE	Total blind	Hereditary cause	Venereal cause	Injury	Definitely preventable
I. Receiving Pensions	135	32	16	23	62
(*a*) Reliable clinical information	84	29	11	17	52
(*b*) Unreliable clinical information	49	2	5	6	9
(*c*) Not now blind	2	1	0	0	1
II. Not receiving pensions	37	8	0	5	8
(*a*) Reliable clinical information	8	6	0	1	6
(*b*) Unreliable clinical information	29	2	0	4	2
III. Total of IA, and IIA	92	35	11	18	58
IV. Total of IB, and IIB	78	4	5	10	11
V. Grand total	170	39	16	28	69

TABLE V

Causes of Hereditary Blindness in Tasmania

DISEASE	Cataract congenital	Detachment of retina	Nystagmus	Leber's optic atrophy	Retinitis pigmentosa
I. Receiving pensions	16	1	1	10	4
(*a*) Reliable clinical information	15	1	1	9	3
(*b*) Unreliable clinical information	0	0	0	1	1
(*c*) Not now blind	1	0	0	0	0
II. Not receiving pensions	6	1	0	1	0
(*a*) Reliable clinical information	4	1	0	1	0
(*b*) Unreliable clinical information	2	0	0	0	0
III. Total of IA, and IIA	19	2	1	10	3
IV. Total of IB, and IIB	2	0	0	1	1
V. Grand total	21	2	1	11	4

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Sociological Problems

The Tasmanian Institution for the Blind, Deaf and Dumb has the following objectives in regard to the blind and deaf in the island of Tasmania.

1. To promote the education, industrial training, employment and general advancement in the life of the blind, deaf, and dumb and their social, economic, and spiritual welfare.
2. To undertake measures for the saving of sight, and the correction of defects of speech.
3. To provide buildings, premises, equipment, materials, and facilities for any such purpose as aforesaid.
4. To co-operate and collaborate with any person or organisation having any similar aims for any of the purposes aforesaid.

The Institution is administered by a Board of Management on which sit representatives of the State Government, the Braille

Writers Association, and Subscribers, and it has entire control of all sociological problems relating to the blind.

The sociological problems relating to sufferers from hereditary eye diseases cover an even wider field, and include the normal sighted, the partially sighted and the blind. I propose dealing with these problems under the following headings :—

1. Education.
2. Employment.
3. Welfare.
4. Prevention.

And to pay special attention to the work undertaken in Tasmania in these directions.

Education

Children and adults with hereditary eye defects naturally fall into five classes as far as educational facilities are concerned.

1. Normal sighted—to 6/12—non progressive.
2. Normal sighted with progressive lesion—*e.g.*, myopia.
3. Partially sighted—non progressive—*e.g.*, nystagmus 6/18 to 6/36.
4. Partially sighted—progressive lesion—*e.g.*, retinitis pigmentosa.
5. Blind—6/60 or less.

(*a*) Primary education in institutions.
(*b*) Higher education.
(*c*) Home tuition.
(*d*) Deaf Blind.

1. Normal sighted.—The main duty of the ophthalmic surgeon is to ascertain that the condition is non-progressive, and the Committee of Enquiry into the Problems relating to partially-sighted children (Crawley, 1934), stresses in more than one page of its report, the necessity for the half-yearly examination of the partially sighted, and I feel this provision should be extended to the normal sighted known to have inherited eye defects, for the above reasons at least. These normal sighted children can be educated along ordinary lines so long as their condition is non-progressive, but should they show signs of deterioration, then the ophthalmic surgeon in charge of the case or school, must recommend the transfer to group 2. On the other hand, hypermetropes after adequate correction and continuous use of their glasses may show a marked improvement in visual acuity, which necessitates the surgeon transferring these cases from group 2 back to group 1.

2. Normal sighted with progressive lesions.—In this group, which chiefly includes the myope, the opinion of an ophthalmic

surgeon of experience is absolutely essential in deciding the merits of each case. The Crawley Report on Partially Sighted Children lays down on page 23 criteria for the special education of myopes.

At the same time on pages 32 and 33 the Report stresses the fact that once the myopia has ceased to increase more than ½ dioptre per annum, and provided the vision and fundus changes are satisfactory, such children should be re-drafted to ordinary schools (group 1). On the other hand, if the malady is rapidly progressive, the child may be forced to enter a blind school in order to complete its tuition. On page 35 the necessity of a bi-annual examination by ophthalmic surgeons of all scholars of partially sighted cases is stressed.

I particularly stress the education of myopes, for at present the educational trend is to divide those with hereditary eye defects into 3 classes, educationally :—

1. Normal sighted.
2. Partially sighted including myopes.
3. Blind.

This is unfortunate, for myopes should not be educated together with the partially sighted as they (myopes) require restricted use of books, and physical exercises.

3. Partially sighted—non progressive.—Group 3 are stationary pupils in the partially sighted class, and their education should run along uninterrupted lines.

4. Partially sighted—progressive.—Group 4 is a difficult one, and just when they should be transferred from the partially sighted class to the Blind Institution must again be decided by the ophthalmic surgeon at his regular periodic examinations. The Crawley Report (1934) on page 25 recommends the education of these patients in " partially sighted " schools as long as practicable. It also devotes the whole of chapter V to weighing the *pros* and *cons* of segregation *versus* non-segregation of the partially sighted, and after a full survey of the merits of each, concludes the discussion in favour of the partially sighted not in schools for such, but in special classes in ordinary schools. The final paragraph to the chapter is as follows :—

" After balancing the advantages of the segregation and non-segregation systems, and recognising fully the difficulties which must be overcome, and the prejudices which must be broken down, the Committee recommends that the education of partially sighted children should be conducted where possible in special classes attached to, and forming an integral part of, the ordinary school. This decision has only been arrived at after studying this system in America, where it is used extensively, and at Liverpool where it has been found most satisfactory."

Myer (1930) in his survey of Sight Saving Classes in the public schools of the United States of America details some of the standards of admission to such classes in America. Variation in the different States is enormous, and the National Society for the prevention of Blindness has given the following guide for finding potential sight saving class (not school) pupils, but they stress emphatically that all cases must be considered individually:

(i) General Statement, children having a visual acuity of 20/70 (Snellen 6/12) or less in the better eye after proper refraction. In addition the following are recommended as potential candidates:—

(*a*) Children in elementary schools having four or more dioptres of myopia.

(*b*) Inactive, subsiding (or regressive) cases, such as interstitial or phlyctenular keratitis, optic neuritis, trachoma, etc., in which some irritation may be present, provided the approval of the attending physician is given.

(ii) All cases must be considered individually.

(iii) Any child who in the opinion of the ophthalmologist would benefit by assignment to a sight saving class, subject to suggestion for treatment and training by such ophthalmologist, and the acceptance of the educational authorities having charge of such classes.

(iv) It is assumed that all the children assigned to sight saving classes have average normal mentality.

5. Blind.—Although group 5 appears straightforward, complaints have arisen in the past, due to the failure of the necessary authorities, to have all candidates for Blind Schools examined by an ophthalmic surgeon before admission. The Crawley Report (1934) describes the education of the partially sighted in the blind institution as " indefensible "—not only does such an education stigmatise the partially sighted, but also it is quite inadequate for their needs in after school life.

(*a*) Primary Education in Institutions.—Schools for blind children have been instituted in practically every country, but in some they have been incorporated with classes for the deaf also. This, to some extent, is unfortunate, as each defect has entirely different problems, and Best (1934) points out that, as in many institutions, the blind are in the minority, their education is subordinate to that of the deaf. But for financial reasons in smaller states and countries his combination is unavoidable although not ideal. It must be remembered that Best (1934) states that it costs

eight times as much to educate each blind child in a residential institution as it does to teach each sighted child in a day school. For transport reasons most blind schools are residential, but the ideal methods of tuition are day schools, which can only function satisfactorily in larger cities. Further, schools for the blind should be as far as possible treated as educational rather than charitable institutions, for to stigmatise the education of the blind as charity is not in accord with the trend of modern thought. Education, nevertheless, should be compulsory for at least 9 years, *i.e.*, from 7-16 years of age; and if higher education is desirable and desired, it can be undertaken on a voluntary basis.

(*b*) Higher Education.—This group contains those who through special adaptability qualify for higher education. This can at first be undertaken at High Schools, and later at a University. The number of blind, able to pass the necessary examination even with the aid of a sighted coach and Braille writer is extremely limited.

(c) Home tuition.—This group is usually carried out by the Welfare (or Field) Officers, and is almost totally confined to adults whose blindness has supervened after school life, and who wish to learn Braille or Moon. For the elderly blind the Moon type appears to be indispensable.

(*d*) Deaf Blind.—This group can seldom come within the scope of this paper, and when some hereditary blind child does become afflicted with deafness it is wise to educate him with the blind rather than the deaf.

Local Conditions

In the Commonwealth of Australia there are 6 States, and in each state capital a blind school is situated, primarily for the education of blind children, but also catering for a limited number of partially sighted. In no part of the Commonwealth has any attempt been made to organise separate classes or schools for the partially sighted or myopes. This I have been at pains to verify by letters addressed to each blind school within the Commonwealth.

At present in Tasmania I am making a determined effort, with the aid of the Director of Education, to establish at least one class for the partially sighted, both in Hobart and Launceston; and a rough survey of metropolitan schools in Hobart is being made to this end. But unfortunately the present government is not sympathetically disposed to the project, and the matter may have to be dropped. It might be mentioned here that the routine examination of the eyesight of school children within Tasmania

is at present carried out by the school nurses, and the survey mentioned above proves conclusively that many children with poor eyesight attending school have never received attention either from an oculist or optician. Both the lack of expert medical supervision and the lack of appropriate treatment is greatly to be regretted.

In 1906 compulsory schooling for the blind and deaf in Tasmania was introduced by legislation, and 7-16 years defined as the appropriate span of tuition for those so affected. It is now felt that if these afflicted children could commence their education at an earlier age, better progress would be made. But further legislation must be enacted to bring this about, and at the moment the government is not disposed to act.

A residential school at the Tasmanian Institution for the Blind caters for these children, but happily to say there are now only six, four of whom have hereditary blindness, attending the school from the whole of Tasmania—a most gratifying fact. The cost of teaching the children is undertaken by the State Education Department, while the State Government contributes £18 per annum per head for maintenance and board. Parents occasionally contribute towards their children's upkeep, but none to a greater extent than £26 per annum.

Higher education is also in force in Tasmania. Two boys, aged 16 years, are at present attending the State High School, and are showing very creditable examination results. While another young man, having matriculated, has commenced his Arts course at the University of Tasmania, with a view to becoming ultimately a teacher of the blind. The High School education is free, while the Tasmanian University has waived the collection of fees in the individual case mentioned.

Home tuition is also carried out extensively by the Welfare Officer, and by adults who have been blind from childhood, and is proving most satisfactory.

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Employment

Persons suffering from hereditary eye diseases should be classified as regards employment, under four headings:—

(1) Normal sighted, (2) Myopes, (3) Partially sighted, (4) Blind, and will be dealt with in this order; with special references to conditions pertaining thereto in Tasmania.

1. Normal Sighted.—Except in rare instances such as the colour blind, these people can avail themselves of any offered occupation. The modern use of red and green signal lights precludes the colour blind from aerial, marine, and transport pursuits, and from certain branches of the textile industry.

2. Myopes.—Nothing has been done for myopes in regard to employment, either in Tasmania or Australia, but when a move is made in this direction within the Commonwealth, the investigations of the Board of Education Committee on the Partially Sighted (Crawley Report, 1934), should not be lost sight of in this regard. The Committee of Enquiry found that the myopes tutored in special classes deteriorated more rapidly in after school years than their fellow myopes tutored in ordinary schools, and they suggest that it appears important that dioptre saving must be continued in after school years to be effective. Consequently occupation should be found for myopes which does not entail lengthy periods of close work, or much bending or straining.

3. Partially Sighted.—With regard to the partially sighted in England some organised attempt has been made to find suitable employment for those people after they leave the sight-saving classes, but so far the result is disappointing. In Australia and Tasmania there are no sight-saving classes, and no attempt has been made to place these people in suitable occupations such as distributing trades, farm work, gardening, canvassing, and domestic duties. Consequently they find the most remunerative occupation for which they are capable, without respect to suitability, this is most regrettable and certainly needs rectifying.

4. Blind.—The employment of the blind has been a most difficult problem ever since Valentin Hauy first instituted it in Paris in 1748. There appear to be two main difficulties. First, to find suitable employment, and secondly to dispose of the articles manufactured at a profitable rate; at the same time it must be remembered that on account of mental, physical or temperamental disabilities at least 65 per cent. of the blind are unemployable (League of Nations Report, 1929). Best (1934) estimates that only 7·7 per cent. of the blind in the United State of America are self supporting. Since the War, many countries have attempted to find fresh employment for sightless workers, and in some instances

blind men and women have been incorporated in sighted teams, working in industrial concerns of various types. For instance, in England (League of Nations Report, 1929), 5 girls work in a team at Messrs. Cadbury, Fry and Pascall's factory packing chocolates; and another 5 at Messrs. Wells, Ltd., on tin toys; and certain processes in soap factories have been declared suitable for blind workers. In Germany this branch has been developed to an extraordinary degree, and 218 occupations have been found amongst sighted workers. On the other hand it is only natural that many employers have avoided utilising the blind in their factories on account of the fear of accident, and the resulting consequences under the various Worker's Compensation Acts. In Tasmania no attempt has been made in this direction, nor have the potentialities of subcontracting (N.L.B. Bulletin No. 1) been investigated to any extent.

In Australia the occupations of the blind are almost entirely confined to broom making, brush making, mat making, piano tuning and music. In Tasmania there is one workshop at the Tasmanian Institution for the Blind, where brush, mat, and basket making are the predominating industries. At present 29 blind men, and 3 blind women are employed there, and the articles manufactured are sold in their own city shop. During the past financial year, articles to the value of £4,790 were sold, being an increase of £800 over the previous year. Each blind operative, besides receiving a blind pension of 18s. per week from the Commonwealth Government, has his or her wages augmented by the Institution, so that each single operative receives in all £2 18s. 0d. per week, and each married operative £3 3s. 0d. per week. During the year under review the factory showed a profit of £180, but as £1,640 had to be paid in augmentation the profit was turned into a loss of £1,460.

Besides this the Welfare Officer of the Institution and her Assistant instruct the blind women in cooking, towel hemming, tatting, knitting, crochet and bead work, from the sale of which they make a few shillings profit each week. So far weaving has not been introduced into their activities.

Music and piano tuning also find employment for a few blind men, but the advent of the radio has greatly reduced the number of pianos requiring tuning; and the advent of the talkies has thrown a great many professional musicians out of employment. This is most unfortunate as these two occupations are probably the most remunerative available for the blind. Here again the scope of employment for the blind is extremely limited, and an enquiry into possible new trades for the blind is being carried out in Australia at the moment. Gardening has not been systematically attempted in Tasmania, but has been taught to several

blind children and adults, in view of the satisfactory results obtained since 1920 by the Guild of Blind Gardeners of Great Britain (N.I.B. Bulletin No. 3). Massage courses have not been instituted in Tasmania and therefore are not available for the blind.

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Welfare

The welfare of the blind in Tasmania is undertaken by the Tasmanian Institution for the Blind, Deaf and Dumb, and closely follows that undertaken in other parts of the world. It is arranged under the following headings:—

(1) Children of pre-school age, (2) Children from 7-16, (3) Adult Blind (*a*) Employable, (*b*) Unemployable and Necessitous, and naturally includes those suffering from the various causes of hereditary blindness.

(1) Children of pre-school age are visited by the welfare officer as occasion arises, and instructions are given to parents along orthodox lines.

(2) Children from 7-16. These are educated in a residential school attached to the Institution, and education is compulsory from the age of 7-16. Like the public schools, 3 terms a year are arranged and the children return to their homes for the vacations.

Any pupil showing high scholastic attainments is specially tutored for the qualifying examination of the State Education Department, the passing of which examination makes the pupil eligible for a High School Education. Later they may qualify for University entrance.

(3) Adult Blind.—At the present time there are 135 persons receiving blind pensions in the State of Tasmania, and of these, 29 men and 3 women are employed in the factory attached to the Institution where brush, mat and basket making are undertaken. The remaining are unemployable to a great extent, or are engaged in minor pursuits, such as cooking and sewing. This is comparable to the figures in England and Wales, where about 70

per cent. of the blind are unemployed owing to age, mental weakness or other defects.

Those not attending the factory are placed under the care of the welfare officer and her assistant, but to date there is no residential home for either old men or old women, as it has been found more satisfactory to find lodgings for them in the homes of the sighted. Best (1934) considers that such special homes should only be instituted as a last resource, and on the whole are objectionable.

Certain facilities are provided for the adult blind either by the State or Commonwealth Governments, State Municipalities, the Tasmanian Institution for the Blind, or by other sources.

(*a*) Free tram passes by the City Councils of Hobart and Launceston.

(*b*) Free railway passes by the State Government. These allow each blind person and guide to travel for the fare of one. In special instances free passes for the blind travelling alone are issued.

(*c*) Free theatre tickets by theatrical and motion picture companies.

(*d*) Free radio licences by the Commonwealth Government.

(*e*) Radio sets either free or at cost price, by the Institution for the Blind.

(*f*) Pensions have been paid by the Commonwealth Government since 1910. These are at the rate of 18s. per week (£46 16s. 0d. per annum) for all blind adults over 16 years of age, whose total income from pension and other sources does not exceed £221 per annum. Re-examination is seldom undertaken, so that undoubtedly certain pensioners who have received benefit from treatment still retain their pensions despite the fact that they are able to earn a livelihood.

(*g*) Augmentation of wages and pensions by the Tasmanian Institution has been mentioned under employment. It suffices here to say that each single operative receives in all £2 18s. 0d. per week, while married operatives receive an extra 5s. per week.

(*h*) Retiring allowances by the Tasmanian Institution. On a blind operative reaching the age of 65 he or she is retired from the factory and a grant of 12s. 6d. per week is made from the Institution funds towards his or her upkeep, in order to supplement their pension of 18s. per week.

(*i*) A Medical Union supervised by the Tasmanian Institution. All blind pensioners by contributing 8s. per annum to the fund receive all medical consultations, and all medicine free —also dental attention at cost price.

(*j*) Braille Writers' Association. Members of this association which works under the auspices of the Institution, take responsibility for all transcribing, importation of literature, training of Braille writers, supervision of Braille Library, and other social activities.

(*k*) Braille Mutual Progress Society also under the aegis of the Institution takes responsibility for weekly social gatherings, which include lectures, card evenings, play readings, and dances. Also visits to museums have been experimented with.

(*l*) Library and talking Books. The former is controlled by the Braille Writers' Association and financed by the Institution. (Free postage of books is permitted by the Commonwealth Government.) Two of the latter have been purchased by the Institution but are not yet to hand.

From the above account it will be seen that every effort is being made in Tasmania to cope with the problems appertaining to the welfare of the blind, and it is hoped before long, that the Institution's activities will be broadened to cover the partially sighted also.

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Prevention of Hereditary Eye Disease

Blindness throughout the world has lately so concerned social workers and Governments that International Associations for the Prevention of Blindness and against trachoma have been set up, as well as much legislation passed to check its apparent increase. As a result of this legislation, blindness from smallpox and ophthalmia neonatorum has greatly declined, while the ravages of trachoma have been slightly checked. But little attempt has been made to check the enormous disability of hereditary eye disease, probably because of the lack of reliable data. In Tasmania I find that 37·7 per cent. of the blind are suffering from hereditary diseases of the eye, while Miles Bickerton (1934) estimates that

24 per cent. of the blind of Great Britain suffer from hereditary eye disease, *i.e.*, 1/4 of the total blind, and he suggests possible remedies. Who, on reading this list of remedies can honestly suggest that more drastic legislation is not long overdue? Bickerton asserts that our present outlook is inhuman—with which I agree—but the difficulty is to persuade the public that reform is urgently necessary. Most men and women approach the subject of heredity in two ways, according to Eldon Moore (1934), either as " a mysterious tricky affair responsible for all those queer things in men, which cannot be explained otherwise, or else it is looked upon as a sort of fatalistic demon whose existence all nice minded people should ignore "; and Eldon Moore continues, " Actually the hereditary mechanism is the most important part of all living things." I feel that very few have grasped the truth of this last sentence.

Franceschetti, before the 1935 meeting of the International Association for Prevention of Blindness, suggested the following measures for diminishing the incidence of hereditary blindness :—

(*a*) Collection of precise and complete statistics.

(*b*) Training of physicians (especially ophthalmologists) in genetics, and the education of authorities and the public.

(*c*) Extension of facilities for pre-marital consultation, and the general introduction of pre-marital certificates.

(*d*) Increased use of social workers.

(*e*) Decrease in consanguineous marriages.

(*f*) Decrease of the transmission of hereditary eye disease, by making sterilisation available to the patient.

No one can fail to agree with every one of the above suggestions.

Let me discuss each of these suggestions, and especially the last suggestion of sterilisation, for its effects would be more far reaching than any of the others.

(*a*) Collection of precise and complete statistics.—With regard to this recommendation, the Report on the Prevention of Blindness (1936), repeatedly stresses the fact that uniform certification of Blindness is essential before dependable statistics can be compiled. It suggests four improvements in this direction, namely :

(1) A universal form for examination and report on all blind persons admitted to the register, and it published in detail the form recommended by the Ministry of Health (1933) in their circular number 1,353.

(2) A Universal Definition of Blindness, and it quotes the Ministry of Health's Circular Number 1,353 which includes such a definition.

(3) The Certification of the blind should be undertaken only by a " medical practitioner with special experience in ophthalmology," and it quotes the Ministry of Health's definition of such a term in their circular number 1,353.

(4) The collection of such statistics by a select sub-committee authorised by the Ministry of Health, and the Board of Education is essential. Authority to form such a Committee and to tabulate the statistical findings, has been given to the Prevention of Blindness Committee.

Within the Commonwealth of Australia, which includes Tasmania in its certification laws, there is absolutely no attempt to compile reliable statistics of the causes of blindness. No form other than that for invalid pensions in general is provided; no detailed definition of blindness has been formulated, and no statistics are kept of the certified blind. Further, the services of experienced ophthalmologists to certify the blind, are not obligatory, but the Commonwealth Pensions Department is now taking the necessary steps to have this rectified.

(*b*) Training of physicians in eugenics, and education of authorities and public.—The Prevention of Blindness (1936) report is emphatic that the training of medical men in ophthalmology is inadequate, and recommends the close attention of the General Medical Council to this fact. The training of medical practitioners, and the education of authorities, and the public in general in genetics has been advocated for many years by the Eugenic Society, but has enlisted little support. Obviously man does not wish to face facts where reproduction of his species is concerned.

(*c*) Extension of facilities for pre-marital consultation, and introduction of pre-marital certificates.—Like sterilisation to be discussed in detail later, this recommendation will obviously receive the whole-hearted disapproval of the populace. Yet a stand must be made sooner or later to combat the evil results of the propagation of hereditary disease. But just where to start concerns every social worker. I think it was Bernard Shaw who said " First choose your parents." It certainly appears to many (and also to me) that some public body should actually choose our parents. Men and women should not be allowed to mate with less respect to eugenics than is shown in breeding of domestic animals. Every couple intending to marry should be forced to appear before a tribunal, present their pedigrees for scrutiny and approval, before marriage is allowed.

Legislation of this type is already in vogue in Roumania, but in no other country has it been attempted up to the present. Those rejected should be advised of the reasons, and suggestions given for treatment. If the reasons for rejection are hereditary

disease, then means should be taken to prevent rejected persons from breeding.

(*d*) Increased use of Social Workers.—In this regard much excellent work has been done in the past, but there is gigantic scope for enlargement in the near future, especially in regard to out-patient work in special hospitals, and general hospitals with large ophthalmic departments.

At present in Tasmania except for the Welfare Officer attached to the Tasmanian Institution for the Blind, there is no Social Worker or Almoner attached to any of the large public hospitals or institutions. Some three years ago one trained Almoner was appointed to the Hobart General Hospital, but after twelve months excellent work, her appointment was terminated and the office classed as unnecessary.

(*e*) Decrease in consanguineous marriages.—Later in this work where consanguinity is discussed in conjunction with nystagmus, optic atrophy and retinitis pigmentosa, it should convince the most ardent sceptic that consanguineous marriages are ill-advised, and should be legislated against at once. Best (1934) considers that " the proportion of the blind with blind relatives is over three times as great among those whose parents are cousins, as it is among the blind as a whole." Surely this is sufficient.

(f) Sterilisation.—At the end of 1933 the following countries throughout the world had passed sterilisation laws:—

26 States of the United States of America—principally in the West. 24 of these States have compulsory laws, and 2 have voluntary ones.

2 States of Canada, both of which have voluntary laws.

1 Canton of Switzerland.

Germany, where sterilisation is compulsory.

Denmark, with a voluntary law.

Except in the case of Germany, none of the above mentioned countries enacted laws on sterilisation to deal with hereditary blindness, but in the main they deal with mental cases and feeble-mindedness.

None of the six States of the Commonwealth of Australia has made any move in the direction of laws for human sterilisation, with the exception of Tasmania, but an inquiry into the subject under discussion should be a matter for investigation by the Commonwealth Government and not by States individually.

The Broche Report on Sterilisation (1933) was very guarded in its recommendations, and suggested very limited legislation to deal with the propagation of the unfit. For many reasons it was opposed to compulsory sterilisation, but strongly advocated the voluntary sterilisation, not only of the mentally unfit, which was

its limited reference, but also certain forms of hereditary blindness, deafness, haemophilia and brachydactyly, etc. This I feel certain will never be satisfactorily accomplished unless the physical results are expounded to the public in clear straightforward terms.

Hereditary diseases of the eye have always interested the eugenists particularly in view of the certainty of diagnosis in experienced hands. The Broche Report of the Departmental Committee on Sterilisation (1933) found its enquiry into hereditary mental deficiency much embarrassed by the fact that there were so many degrees of mental deficiency, and so many borderline cases. This is not so with hereditary eye diseases which are clear cut, and well recognised diseases. Yet throughout the whole Broche Report (1933) there is only one practical instance mentioned (page 43) of the sterilisation of the hereditary blind. In view of the fact that so much has been done to prevent blindness in other directions, surely this new avenue of prevention should be utilised without delay.

On the subject of the legality of sterilisation, the above reports are quite emphatic. Legality of therapeutic sterilisation is not disputed, but the legality of eugenic sterilisation in the present state of the law in England is couched in the following terms on page 6, " but in practice it appears to be almost universally accepted that eugenic sterilisation is illegal, and involves the surgeon concerned in the risk of legal proceedings, even though the full consent of the patient has been obtained." On the other hand Beasley (1935) discussing the legality of the sterilisation of the unfit in Western Australia asserts, " Sterilisation performed at the request of a patient who understands the nature and result of the operation, and freely consents thereto, does not constitute a disablement within the meaning of section 294 (of the West Australian Criminal Code), and does not involve in criminal responsibility the surgeon who performs the operation."

H. C. Lewis, barrister at law, in a private communication in reference to the laws of Tasmania in regard to eugenic sterilisation, is of the opinion that it is inadvisable in this island under the existing laws.

If clear thinking men and women with inherited defects commence to ask for sterilisation, as has been the case in Tasmania, then the time is more than ripe for the Government of this Commonwealth to take the matter up, and have the proper legislation for voluntary sterilisation enacted without delay, to include not only the mentally unfit, but those suffering from all inherited diseases.

If this is enacted there is then a possibility of stamping out over half the preventable blindness in Tasmania. A most pleasing prospect.

The words of Byron seem appropriate here:—

" Hereditary Bondsmen ! know ye not
Who would be free, themselves must strike the blow?"

—*Childe Harold*, 1-86.

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(To be continued)

USE OF CONTACT GLASS FOR COSMETIC PURPOSE

BY

S. K. MUKERJEE, F.R.C.S.(Edin.), D.O.(Oxon.), D.O.M.S.(Lond.)

PROFESSOR OF OPHTHALMOLOGY, CARMICHAEL MEDICAL COLLEGE, BELGACHIA, AND HONORARY OPHTHALMIC SURGEON, CALCUTTA MEDICAL COLLEGE HOSPITALS

THE indications for the use of a contact glass besides its use in the various forms of ametropia are recorded in the literature. In the March 1931 issue of the *Lancet* in the paper " on the use of Contact Glasses " by Leopold Heine, it was noticed that contact glasses might be used for cosmetic purposes. As illustrative cases of the above indication did not come to my notice, I venture to publish the following case of mine which proved to be very successful.

The patient a girl aged 16 years, was brought to me from Patna just before her marriage to tattoo the cornea of her left eye which became leucomatous as a result of small pox in her early childhood. The whole idea was to conceal the disfiguration. It is needless to mention that the parties were unwilling to have the ball removed.

From the history it was learnt that attempts had been made twice, once in Calcutta and once in Bombay to darken the cornea by a tattooing operation. But the operations in both places were unsuccessful. On examining the patient one could easily find that it was not a suitable case for further tattooing as the cornea was very thin, and the iris tissue was entangled in the wound, and the cornea was slightly staphylomatous.

Failing to help the girl with operative interference it was decided to utilise the effect of a contact glass for cosmetic purpose. Fortunately 2/8 size fitted her quite nicely and she was gradually trained for a few days to insert and take out the contact glass herself, which she could do easily after three days. The contact glass was nicely painted inside with an oil painting to match the colour of the other eye and the result was marvellous, as shown in Fig. 2.

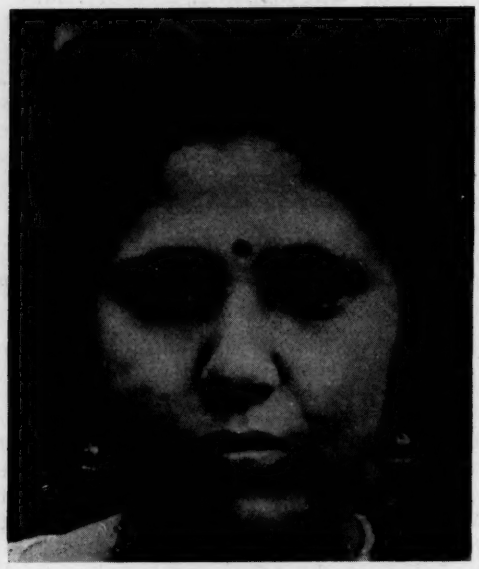


FIG. 1.

Before the insertion of contact glass.

FIG 2.

After the insertion of painted contact glass.

The two photos clearly indicate the difference of the cosmetic effect and it is hoped that the publication of this case of mine will draw the attention of ophthalmologists to the cosmetic use of contact glasses.

STENOPAEIC SPECTACLES

BY

A. L. YOUNG

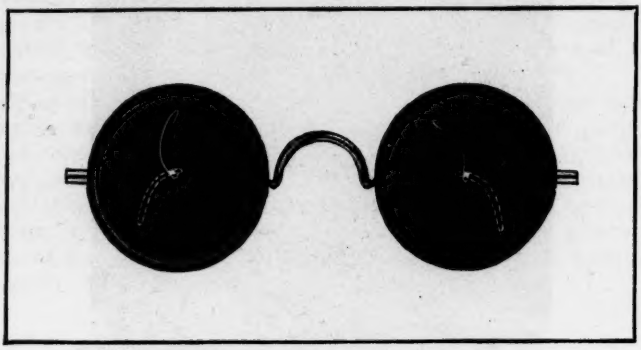
LONDON

THE illustration shows a type of stenopaeic spectacle designed to overcome the disadvantage of present forms, in which the apertures are fixed in position, and may not be at the requisite interpupillary distance of the patient for whom they are required.

After diathermy operations for detached retina, when stenopaeic spectacles are worn, it is important, if the eyes are to be kept at rest, for the interpupillary distance to be correct for that particular

patient, and after certain operations for squint it may be of advantage to be able to vary the relative position of the apertures for different patients.

In this new type there are two discs back to back which can be rotated independently of each other. Each disc is perforated by a curved slit, the aperture being produced by the overlapping of the slits. By a suitable manoeuvre, it is possible to place the aperture in any position on the disc save in the exact centre, an



apparent disadvantage which can be overcome by de-centring an equal amount in each disc. Thus a vertical as well as a horizontal correction may be made. It is also possible, of course, to obliterate the aperture should only one be required.

The slits are at present made 2 mm. in width. Rotation is facilitated by lightly smearing the opposing surfaces of the discs with vaseline; they are sufficiently firmly gripped by the rims to prevent accidental movement.

The spectacles have been made by, and are obtainable from, Messrs. Clement Clarke of Wigmore Street.

ANNOTATION

Blindness in Great Britain

Some interesting figures were supplied by the Minister of Health in moving, in the House of Commons, the second reading of the Blind Persons Bill on November 11, 1937. He stated that the total number of blind persons registered in England, Scotland and Wales was about 78,000. Of these approximately 57,000 were aged

50 years or more, and there were 8,000 persons between the ages of 40 and 50 years. The increase in the total number of the blind was not, in his opinion, due to any real increase in numbers, but to more complete registration, longer life, and increased inducements to blind people to register at the present time. The total number of blind children between the ages of 5 and 16 years had fallen since the year 1925 from 3,104 to 1,916. Only 2·95 per cent. of the 8,700 new cases which were registered in the year ending March 31, 1937, were under the age of 16 years, and about 89 per cent. were over the age of 40 years.

As is well known, the first clause of this Bill provides for a reduction of the age for the receipt of old age pensions in the case of blind persons from 50 to 40 years of age.

No one will grudge the blind the amelioration of their lot which is fore-shadowed in this Bill, if, as we all hope, it passes on to the Statute Book.

The age of 40 years was chosen because of the difficulty of teaching a trade after that age. A lower limit would diminish the incentive of blind persons to attempt to earn their daily bread.

The only regrets that we can see will be those of the blind who have gone before. We fancy we see "Poluphloisboisterous" Homer congratulating the first of the new beneficiaries under the Act who is conveyed across the Styx and commenting on how times are changed. He never received 10 drachmae a week.

ABSTRACTS

I.—OPTIC NERVE

(1) **Behr, Carl (Hamburg).—New anatomical facts on papilloedema. (Neue anatomische Befunde bei Stauungspapille).** *Arch. f. Ophthal.*, Vol. CXXXVII, p. 1.

(1) In this contribution to the pathogenesis of papilloedema of intracranial origin **Behr** demonstrates in anatomical preparations, embedded in gelatine so as to avoid shrinkage and the formation of artefacts, the presence of spaces or small cavities between the nerve fibre bundles along the whole length of the optic nerve from the disc to the optic canal: the nerve fibres separated from each other or pressed together in such a way as to indicate that these spaces must have been caused by a collection of fluid under pressure between the nerve fibres *intra vitam*. He lays emphasis on the fact that this oedema is intrafascicular, and is not present in or under the pial sheath, septa or in the axial connective tissue strand;

the oedema causing the swelling of the papilla has no relation whatever to the central vessels of the disc or their perivascular tissue.

The distribution of the spaces in the optic nerve, which roughly corresponds with the defects in the visual fields, depends on the duration and intensity of the papilloedema; in the initial stages they appear only in the nerve fibres immediately under the pial sheath, but as the process spreads they extend more and more towards the axis of the nerve and eventually throughout it.

This condition, according to his views, is one of stasis due to obstruction to the flow of the fluid in the nerve fibres from the papilla towards the cranial cavity by the increased intracranial pressure. It is also seen in extra-neural tumours of the orbit.

It is contended that the anatomical facts here described disprove the theory that the subarachnoid fluid is under increased pressure and presses along the perivascular spaces of the central vessels into the optic disc; there is no evidence of obstruction to the blood flow in the retinal vein in its passage through the subarachnoid space, while the changes in the pressure and diameters of the central vessels, which have been cited as the cause of papilloedema, are easily explained by a stasis of the intrafascicular fluid.

THOS. SNOWBALL.

(2) **Sobanski, J. (Warsaw).—The value of dynamometric investigations in determining the pathogenesis of papilloedema. (Der Wert dynamometrischer Untersuchung für die Erklärung der Entstehung der Stauungspapille).** *Arch. f. Ophthal.*, Vol. CXXXVII, p. 84.

(2) **Sobanski's** researches on papilloedema were partly clinical, in which he made dynamometric estimations of the pressure in the central retinal vessels and a comparison of the blood pressure with that of the cerebro-spinal fluid: partly experimental, to discover by compression of the retrobulbar portion of the optic nerve the position in the nerve at which the action of the subarachnoid fluid on the blood pressure in the central vein takes place. From his experiments it was shown that papilloedema developed only on compression of the part of the nerve containing the central retinal vessels.

His investigations by dynamometric measurements on the disturbances of the retinal circulation in increased intracranial pressure proved that papilloedema does not supervene until the ratio of the pressure in the central vein to the diastolic pressure in the artery exceeds 1:1·5, the swelling of the papilla increasing with further rise in the ratio.

This disturbance in the normal relation of the pressure in the central vein and the artery is the most important factor in the

pathogenesis of papilloedema, and is due to obstruction to the circulation in the central vein from the eye, this obstruction taking place in the part of the nerve carrying the central vessels.

Of secondary importance is the obstruction to the return flow of the tissue fluid from the eye along the optic nerve, while the state of the intra-ocular pressure, the size of the papilla and other factors have an indirect effect.

This problem can be solved only by dynamometric estimations added to experiment, not by pathological-anatomical investigations alone.

THOS. SNOWBALL.

(3) **Much, Viktor (Lucerne).—Amyl nitrite in the treatment of diseases of the optic nerve. (Das Amylnitrit in der Behandlung von Erkrankungen des Opticus).** *Zeitschr. f. Augenheilk.*, Vol. LXXXIX, p. 58, 1936.

(3) **Much** reports favourably on the effects of amyl nitrite in five cases of toxic amblyopia (tobacco and alcohol), one case of retrobulbar neuritis in disseminated sclerosis, one case of renal neuro-retinitis and in one case of post-neuritic atrophy. The amyl nitrite was inhaled in doses of 2—4 drops. In five cases of toxic amblyopia and the case of post-neuritic atrophy recovery was complete. The two other cases were greatly improved.

ARNOLD SORSBY.

II.—RETINA

(1) **Messinger, Harry C. and Clarke, B. Earl (Providence, R.I.). —Tumours in the tuberous sclerosis.** *Arch. of Ophthal.*, July, 1937.

(1) Retinal involvement in tuberous sclerosis was first described by van der Hoeve in 1921, and the disease itself by Bourneville in a series of articles published from 1880 to 1898. It consists in a rare form of cerebral sclerosis, associated during life with mental deficiency, epilepsy and adenoma sebaceum, distributed in butterfly fashion on the face. The syndrome is variable however, and there may be multiple tumours of mixed undifferentiated cells, in the heart, kidney, spleen and eye, as well as in the brain. A family history of developmental defects and psychopathic trends is common. The retinal tumours occur as oval or circular, white or grey areas, with an average diameter of about one-half that of the disc. The tumours are occasionally larger and nodular, extending into the vitreous usually at or near the disc. In the brain the typical,

multiple small tumours are in the cortex, but there are often others, projecting into the ventricles and sometimes causing papilloedema. **Messinger** and **Clarke's** case occurred in a man, aged 20 years, who had had convulsive seizures since the age of four, was afflicted with adenoma sebaceum and died from broncho-pneumonia. Ophthalmoscopically, his right eye was found to contain a white nodular tumour extending forwards 5 D. and obscuring the disc. Post-mortem there were multiple tumours of the brain, rhabdomyoma of the heart, and lipomata of the kidneys and adenoma sebaceum. The tumour in the eye involved all the layers of the retina and invaded part of the optic nerve. Its central portion was irregularly ossified. The cells of the tumour varied in size and shape, and their boundaries were indistinct, suggesting a syncitium. The sections were examined by Ida Mann who suggested that the tumour had arisen during stage 2 of retinal differentiation (from the sixth week to the third month) from glia cells of the inner neuroblastic layer, with a few undifferentiated amacrine and ganglion cells included. The authors conclude from these observations and the descriptions in the literature, that the growths in the brain and other organs are haematomata rather than true tumours.

F. A. W-N.

(2) **Scheyhing, H. (München).—Cure of a glioma retinae by X-rays. (Heilung eines Netzhautglioms durch Röntgenbestrahlung).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 756, 1937.

(2) **Scheyhing** describes the successful treatment of a retinal glioma by X-rays in increasing doses over several years. A cataract developed which was removed and the patient retained good vision. The companion eye had been removed for advanced glioma retinae. Details of dosage are given.

D. R. CAMPBELL.

(3) **Vogt, A. (Zürich).—Histological findings in another case of relatively recent (senile-myopic) retinal detachment. (Histologischer Befund eines weiteren Falles von relativ frischer spontaner (senil-myoper) Netzhautablösung).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 735, 1937.

(3) **Vogt** describes the histology of an eye of a patient, aged 72 years, who died 37 days after the onset of a retinal detachment. A previous clinical examination had shown a hole at the periphery and an operation had been performed which had not succeeded in closing the hole. There was marked thinning of the retina in the region of the hole and the torn away piece appeared to have been pulled off by the neighbouring vitreous to which it was still adherent.

Although the eye was myopic the author considers that the centripetal action of the vitreous was the primary cause of the detachment, since the whole of the vitreous was massed forwards close behind the lens. There was no evidence of inflammatory changes in the vitreous.

D. R. CAMPBELL.

III.—CORNEA

(1) **MacMillan, J. A. and Cone, Wm. (Montreal).—The prevention and treatment of keratitis neuroparalytica by closure of the lachrymal canaliculi.** *Canadian Med. Assoc. Jl.*, October, 1937.

(1) **MacMillan** and **Cone** read this paper at the 30th meeting of the Society of Neurological Surgeons, at Montreal, on May 31, 1937, and at the meeting of the Canadian Medical Association, at Ottawa, in June, 1937. They point out that the present methods of treatment of neuroparalytic keratitis, though effective, are cumbersome. Their studies have convinced them that although the cornea is anaesthetic it is the diminished tear secretion which is the primary factor in these corneal lesions.

They report a case where a satisfactory result was obtained by closing the canaliculi and maintained over a period of five months, with incidentally an improvement in vision from 6/30 to 6/15.

The canaliculi were closed by electro-coagulation. Sensation being lost, no anaesthetic is required; but the canals tend to become patent again after a few days. When the canaliculus is slit with the actual cautery it is closed permanently.

A bibliography of nine items is appended.

R. R. J.

(2) **Jese, L. (Ljubljana).—Keratitis nummularis. (Ueber Keratitis nummularis).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVI, p. 219, 1936.

(2) **Jese** discusses 102 cases of keratitis nummularis he has seen since 1932, mostly in agricultural workers. On an average the number of lesions is 10 to 15; only four patients had a single nodule, and seven two nodules. The solitary nodules were always peripheral. In size the nodules ranged from being first visible with oblique illumination to round areas 3 mm. in diameter; the peripheral nodules were always the largest. There was some plan in their arrangement and they were situated in the anterior third of the parenchyma. They were not sharply demarcated and the

slit-lamp revealed the characteristic white dots in the epithelium. The fresh lesion pushes the epithelium forward; later on this flattens out leaving a glistening, facetted surface with a yellow centre. Superficial vascularisation was common, deep vascularisation and also iritis uncommon, though hyperaemia of the iris was present in 31 cases. There is no treatment besides atropine and symptomatic measures. The author holds that the conditions is allied to the herpes group.

ARNOLD SORSBY.

(3) **Vogt, A. (Zürich).—Keratoconus lines and their anatomical basis. (Die Keratoconus Linien der Kegelspitze und ihre anatomische Grundlage).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 577, 1937.

(3) **Vogt** records his observations, by slit-lamp and histological investigation, on the condition of keratoconus. Three types of opacity are present—the most superficial is in Bowman's layer, the second consisting of striae in the deep parenchyma and the third are tears in Descemet's membrane. The author discusses the origin of these changes.

D. R. CAMPBELL.

IV.—MEDICAL OPHTHALMOLOGY

(1) **Testa (Turin).—Changes of the optic foramina in oxycephaly. (Oxicefalia ed alterazione dei forami ottici).** *Rass. Ital. d'Ottal.*, March-April, 1937.

(1) **Testa** gives an account of a case of advanced oxycephaly which he has followed for some years; when first seen the boy had 6/10 in the right eye; hand movements in the left. The left optic foramen was shown by the radiograph to be reduced to a narrow slit; the right also was not so large as in a normal skull. In 1936 the boy was again examined and the left eye was found to be totally blind, but the right eye still kept an acuity of 6/10.

The author discusses the possibility of surgical interference. The optic canal may be reached either from the orbit or from the cavity of the skull; in either case the roof of the canal must be removed to release the nerve from the pressure. The chief difficulty is that in most cases the nerve has been hopelessly damaged before the condition is recognised as serious, by those in charge of the child.

HAROLD GRIMSDALE.

(2) **Kyrieleis, W. (Hamburg).—Fundus changes in lymphogranulomatosis. (Ueber Augenhintergrundsveränderungen bei Lymphogranulomatose).** *Zeitschr. f. Augenheilk.*, Vol. LXXXIX, p. 183, 1936.

(2) **Kyrieleis** recalls that no fundus changes have been reported in Hodgkin's disease. Reviewing doubtful instances recorded in the literature, he holds that there are no characteristic fundus lesions. This is borne out by a case of Hodgkin's disease reported in full. Fundus changes did appear in this case six days before death. Clinically and histologically these were the changes found in any severe anaemia. The characteristic histological picture of Hodgkin's disease could not be found in the retina or around its blood vessels

ARNOLD SORSBY.

(3) **Prewitt, L. H. (Ottumwa, Iowa).—Retinal detachment due to Allergy.** *Arch. of Ophthal.*, July, 1937.

(3) **Prewitt's** patient was a man, aged 62 years, with myopic astigmatism and some lens opacities. At his first examination he complained of blurred vision in the left eye and was found to have an early retinal detachment. He also had some nodular swellings on his body which he said were due to eating certain food.

Complete rest for two weeks and the application of pads and bandages brought about reposition of the retina. Three months later the condition recurred and a large detachment developed which did not go back. Four months later an early detachment developed in the right eye and was cured by rest, to recur in another four months and again be cured by rest. Two months later still the lens having become more opaque, was extracted intracapsularly and advantage was taken of his being in hospital to apply intradermal tests. He gave positive reactions to a large number of different substances and a diet was planned which omitted the offending allergens with the result that he remained free from attacks over a period of about four years, except on one occasion when a flare up occurred following a "Thanksgiving" dinner at which he ate turkey's liver.

F. A. W-N.

V.—MISCELLANEOUS

(1) **Fuchs, A. (Wien).—Leprosy bacilli in eyes with a normal clinical appearance. (Ueber Leprabazillen in klinisch normal erscheinenden Augen).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 728, 1937.

(1) **Fuchs** investigated the eyes of three patients suffering from leprosy. In life, they had been free from ocular symptoms but histologically the following changes were found:—

1. Inflammatory areas in the episclera, with bacilli in the periphery of the cornea.
2. Numerous bacilli and lepra cells in the ciliary body and ora serrata—but without inflammatory reaction in these tissues.

He considers that the bacilli probably enter the eyeball by the blood stream and give rise to these changes long before the more obvious inflammation of the lids appears.

D. R. CAMPBELL.

(2) **Tertsch, R. (Vienna).—Another case of periarteritis nodosa with changes on the arteries of the extra-ocular muscles. (ein weiteres Fall von Periarteritis nodosa mit Veränderungen an den Arterien der äussern Augenmuskeln).** *Zeitschr. f. Augenheilk.*, Vol. LXXXVII, p. 294, 1935.

(2) **Tertsch** records another case with ocular involvement in this highly fatal infection. In his case a systematic post-mortem examination revealed the typical periarterial infiltration in three of the extra-ocular muscles. In contrast to other reported cases with ocular involvement, the globes themselves were unaffected.

ARNOLD SORSBY.

(3) **Müller, H. K. (Berlin).—The mechanics of the exfoliation of the lens capsule in capsular glaucoma. (Zur Mechanik der Abschilferung der Linsenkapsel bei Glaukoma capsulare).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 653, 1937.

(3) **Müller** describes an interesting case which proves that in capsular glaucoma the exfoliation of the lens capsule is due to the friction of the iris on the anterior surface of the lens. A preliminary iridectomy had been performed on the left eye some time previously. In both eyes there was a greyish white ring of opacity in the anterior lens capsule, which corresponded to the customary position of the edge of the pupil. In the left eye this was absent in the area corresponding to the coloboma. Fine white particles, which were probably lens matter, could be seen adherent to the zonular fibres. The author emphasizes the importance of prophylactic

treatment by the use of miotics or mydriatics in preventing a constant position of the pupil, and suggests that capsular glaucoma may be more common than is usually supposed. Indeed, Hörven found signs of exfoliation of the lens capsule in 85 per cent. of his cases of glaucoma simplex.

D. R. CAMPBELL.

(4) **Streiff, E. B. (Genf).—The action of syntropan on normal and glaucomatous eyes. (Wirkung des Syntropans im normalen und glaukomatösen Augen).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 504, 1937.

(4) **Streiff** describes the action of a 5 per cent. solution of syntropan (Hoffman-la-Roche). It stings the eye slightly and produces maximum mydriasis in 20—30 minutes. Usually the pupil begins to contract again in 45—60 minutes. The effect on accommodation is 0·5—0·75 dioptres and lasts about the same time. The action of the drug was compared with that of 2 per cent. cocaine and 2 per cent. homatropine. It was a better mydriatic than the former and almost equal to the latter. 2 per cent. pilocarpine was found to cause a rapid miosis after syntropan—a significant point in the preferential use of this drug for examining the fundus of an eye suspected of glaucoma. It does not damage the corneal epithelium.

D. R. CAMPBELL.

(5) **Nizetic, Z. (Belgrave). — Experience with cyclotropin in diseases of the anterior portion of the globe. (Erfahrungen mit Cyclotropin bei Erkrankungen des vorderen Bulbusabschnittes).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCVIII, p. 658, 1937.

(5) **Nizetic** gives the result of his experience of the use of cyclotropin in over 400 cases. The drug is made in 5 c.c. ampoules (Schering) and consists of 2·0 gm. urotropin, with 0·8 gm. sodium salicylate and 0·2 gm. caffeine-sodium salicylate. It has been demonstrated that urotropin soon appears in the aqueous humour—whether given per os or intravenously and that it breaks down into formaldehyde which persists for as long as 19 hours. Clinically, cyclotropin is of greatest value in cases of perforating injury where sympathetic inflammation has just begun and in all cases of cyclitis and iridocyclitis. It is best given intravenously, but it can also be given painlessly by intramuscular injection, if the more recent preparation of cyclotropin and novocaine be used. Two cases of herpetic inflammation gave good results, showing that cyclotropin has a good anti-infective action.

D. R. CAMPBELL.

BOOK NOTICES

Der Psychische Restitutionseffect. By Dr. OTTO LÖWENSTEIN, formerly Professor and Director of the Pathopsychological Institute of the University of Bonn. Pp. 92. Basel: Benno Schwabe & Co. 1937. Price, 8 Swiss francs.

Dr. Otto Löwenstein has already published two brochures on the pupil reactions in their relation to the early diagnosis of syphilitic diseases of the nervous system and schizophrenia. The method used has been the kinematographic registration of the pupillary movements in various conditions and under various modes of stimulation. The importance of the pupil reactions in diagnosis and prognosis has been fully recognized—certainly since the epoch-making observation of Argyll Robertson—even though it be an exaggeration to describe the iris as "the swift-footed, golden winged sandal of the gods, and in another sense the link between heaven and earth, *i.e.*, between the body and the Soul," as a writer in the Psychiatrisch-Neurologische Wochenschrift says. It may well be that kinematographic records of the pupil reactions will take a place in the finer diagnostic methods comparable to that of the electro-cardiogram.

The present brochure deals with the recovery of the pupil from prolonged fatigue and from the severe but less prolonged fatigue in diseased states, such as lues. The author is convinced that the partial recovery which is definitely found to follow sensory and psychical stimuli is a true restitution of function, brought about through the mediation of the vegetative nervous system. Similar results are obtained for the tendon reflexes. Another possible explanation would seem to be that it is of the nature of flogging a tired horse. Whatever the interpretation the researches are worthy of the careful consideration of ophthalmologists as well as neurologists.

Introduction to Physiological Optics. By JAMES P. C. SOUTHALL, Professor of Physics, Columbia University. Pp. 426. London, New York, Toronto: Oxford University Press. 1937. Price, 5·50 dollars.

Professor Southall is known to the readers of this Journal as the author of an excellent book on "Mirrors, Prisms, and Lenses," which was reviewed recently in our columns; and also as the editor of the English translation of Helmholtz's Physiological Optics. The present book is an elaboration of lectures delivered to undergraduate students in the Columbia University, New York. It covers the usual ground of such a course of lectures, and, as in the former work already mentioned, is distinguished by the accuracy of the information and the particularly able and interesting manner in which that

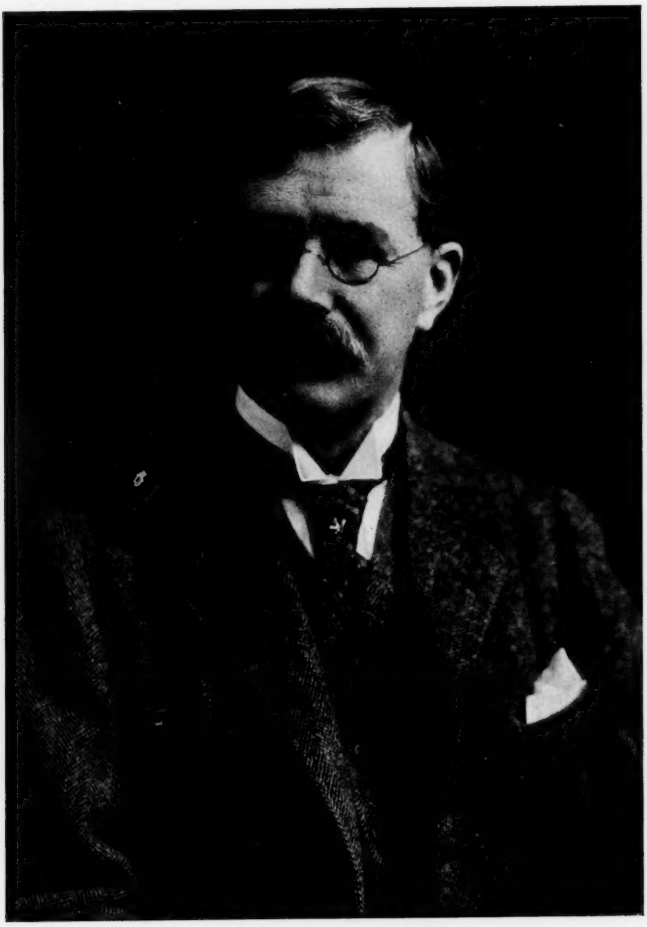


Photo by T. & R. Annan, Glasgow

ERNEST THOMSON

1865—1937

UNIV. OF MICH.

information is conveyed. The chapters deal with the organ of vision; the optical system of the eye; correction eyeglasses, hyperopia, myopia, and astigmatism; movements of the eyeball in its socket; nature of binocular vision; rod vision and cone vision; colour vision and colorimetry; concerning theories of colour vision; and temporal and spatial reactions of the organ of vision. Apart from one or two statements in the anatomical part (*i.e.*, on pp. 13, 15, 21) we have nothing but praise for the book. As might be expected the optical parts are specially good, and the theory of correcting glasses is dealt with in masterly fashion, such modern refinements as "punktal," "katral," and contact glasses being lucidly described and explained.

The book throughout is thoroughly up-to-date, as for example in the explanation of the Pulfrich phenomenon; and should be read even by those who are already conversant with such classics as Helmholtz's treatise, and so on. It is very well printed and illustrated.

OBITUARY

ERNEST THOMSON

IT is with very great regret that we record the death of Ernest Thomson, on November 22, 1937. His health broke down about two years ago and although he made a partial recovery he was left very gravely handicapped and unable to work. He made two gallant attempts to continue his voluntary proof reading for this journal last year, and it was a real sorrow to him to find that he was no longer up to the work, which he relinquished with great regret. What this loss of his work has meant to the journal the Editors fully appreciate. He read our proofs month by month for 19 years for the joy of helping, and his help was recognised by all in the Editorial Department.

William Ernest Francis Thomson, to give him his full title, was born on February 12, 1865, in Edinburgh. His father was William Mann Thomson, Advocate, and his mother was daughter of John White, of Glasgow. His paternal grandfather was Robert Thomson, Writer, in Glasgow.

Ernest Thomson's father died at an early age, when his son was only four years of age. Later his mother married an army officer with a large family. Ernest's early years were spent in Ireland, the Channel Islands and at Chatham. For a very brief period he was at a school in Jersey, but in 1873 he entered the United Services College, Westward Ho!, where he was a contemporary of Kipling. In his recollections he was wont to point out that neither

Greek nor German was taught at this school in his day, but that English was "pumped into" the pupils as much as possible. Especially for "the services examinations, the boys had to write rapidly to dictation in a competitive manner, where a single mistake in spelling would cost many marks." Doubtless this was the origin of his mastery of the English language and his remarkable proficiency in correcting proofs.

On leaving school Ernest Thomson went for a short period into his uncle's office in Glasgow to study for the law, but this proved uncongenial, and he pushed on with his education at Glasgow University, where he took his M.A. in 1885. His friendship with Harry Bamber, whose sister was his first wife, inclined his thoughts towards medicine; and he started in Glasgow, but migrated half way through his course, to Edinburgh, where he graduated M.B., C.M., in 1889. After qualification he spent some two or three years as a ship's surgeon, travelling to the Cape and to India. Though never sea sick, he confessed to being home sick, and he returned to this country in 1890 and married Miss Bamber. His first venture in practice was medical officer to a parish, with private practice, in Orkney. The 1889 epidemic of influenza had visited the island severely, and Ernest Thomson soon found that the practice did not offer sufficient scope, so he sold out, and returning to Glasgow, put up his plate, after a short period of post-graduate work in London, where he first seriously took up the study of ophthalmology. In Glasgow he began work at the Eye Infirmary, where Leslie Buchanan was the house surgeon at that time. They were each shortly afterwards elected to the junior staff. Life was a struggle at this time, as it is with so many of us at the start, and Ernest Thomson put in a good deal of work in the Physiological Laboratory of Professor McKendrick. He obtained the post of lecturer at the Western Medical School, and was also for five years Professor of Physiology at Anderson's College Medical School. He took his M.D. in 1893, and the F.F.P.S.Glas. in 1897. In the following year he became a member of the Ophthalmological Society of the United Kingdom.

In 1902 he was elected to the senior staff of the Glasgow Eye Infirmary. On the foundation of "The Ophthalmoscope" in 1903, Ernest Thomson became editorial secretary and later sub-editor.

In 1911 he paid a long visit to the continental eye clinics and his notes on continental work were published in eight numbers of "The Ophthalmoscope" in 1912. Not long after, he decided to give up his practice in Glasgow for departmental work, and was appointed part time ophthalmic surgeon to the Glasgow School Board, and later, whole time ophthalmic surgeon to the Education Committee of the County of Lanark.

During the War years he was single handed, and had to under-

take the inspection of school children as well as their treatment, all the other medical officers of the County Education Committee having volunteered for service. In 1921 he resigned his whole time appointment, and the Committee, a year later, decided to appoint two part-time officers instead, and Ernest Thomson was glad to come back.

After leaving Glasgow he moved to Bothwell, and in 1923 to Stirling, which was his home for the rest of his life. He gave up operative work in 1914, but in Stirling did some private practice as well as his school work.

Ernest Thomson wrote a great many papers, mainly on ophthalmological subjects. His first important paper, however, was his M.D. thesis, for which he obtained the Gold Medal. It was on "The true position of oxygen as a restorative in carbonic acid poisoning," and was published in the *Glasgow Medical Journal* in 1894. Among other early papers was one in the *Edinburgh Medical Journal* in 1897 on "Sensory Aphasia, with sector-shaped homonymous defect of the Fields of Vision." To "The Ophthalmoscope" he was a regular contributor from the start until 1914. Many of his papers were written conjointly with his friend Dr. Leslie Buchanan. In 1909-10 he was the author of a long review on Detachment of the Retina, and in 1912 his Notes on the Continental Clinics, noticed above, were published.

In the *British Journal of Ophthalmology* he published many papers. He was a member of the executive editorial committee from the start. Altogether more than 66 papers stand to his credit. We have already attempted to indicate the enormous amount of voluntary work he did for us in proof reading. In the matter of composition nothing but the best satisfied him; and we fear that our English composition has suffered in those numbers which have appeared without his kindly criticism. Henderson was always insistent on the value of Ernest Thomson's work for the Journal, and the writer, who has had the privilege of his friendship in correspondence during the past 14 years, can honestly say that the high standard of the composition of the Journal has been due as much to Ernest Thomson as to anyone else. His first wife having died in 1896, he married in 1898 Miss Isabel Wingate, to whom the sincere sympathy of British ophthalmology will go out in her bereavement.

Mr. HARRY HOLMES

On October 30, 1937, aged 63 years, there passed away in the person of Mr. Harry Holmes, B.A., M.B., B.Ch., one of the best friends of the Voluntary Hospital System of Merseyside.

Springing from sturdy yeoman stock in Lincolnshire he had all the good qualities of the typical Englishman, sharing also in the reticence and self-effacement.

Passing through St. John's College, Cambridge, and then through Barts., he qualified at the age of 21, and early in his career became associated with the Wigan Infirmary as junior, and then as senior House Surgeon.

Appointed in 1904 to the honorary staff of the Eye and Ear Infirmary, Myrtle Street, Liverpool, he spent the rest of his life, health and energy in unostentatious hard work for this and kindred medical institutions in Wigan, as ophthalmic and aural surgeon.

A brilliant Chess player, who held many championships, he nevertheless put his duty to his work first; indeed, there were few who maintained a more consistent level of all round good results in his clinical work year in, year out.

Joining the North of England Ophthalmological Society in 1914, he served on its Council in 1925. His skill and ability often went un-noticed, so modestly was it tendered, but it was on the point of character that he will be best remembered by his medical friends as "one more skilled to raise the wretched than to rise."

NOTES

Appointments

Mr. H. B. STALLARD has been elected Asssitant Ophthalmic Surgeon to St. Bartholomew's Hospital.

Mr. E. F. KING has been elected Assistant Ophthalmic Surgeon to the Westminster Hospital.

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The Treacher Collins Prize

UNDER the above title the Council of the Ophthalmological Society of the United Kingdom has instituted a Prize of £100 awarded Triennially for the best Essay submitted upon a subject selected by the Council.

The Prize shall be open to qualified Medical Practitioners of any nationality.

The Essay shall be written in the English language.

The Subject of the first award of the Prize is "Cerebro-spinal Disease and its Relation to the Optic Nerve."

The closing date for sending in Essays for the first award is December 31, 1938. Essays should be submitted to the Honorary Secretary, Ophthalmological Society of the United Kingdom, 5,

Racquet Court, Fleet Street, E.C.4., from whom also any further particulars can be obtained. No name should be on any Essay, but a distinguishing pseudonym or quotation, which should also be upon a sealed envelope containing the candidate's name and address. This envelope should accompany the Essay.

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Middlemore Prize

THE Council of the British Medical Association is prepared to consider the award of this prize in the year 1939 to the author of the best essay on:—"The underlying causes of glaucoma, including notes on the lines of enquiry which have been pursued, with suggestions as to future research in clinic and laboratory."

In competing for this prize authors must submit essays which must reach the Secretary, British Medical Association, B.M.A. House, Tavistock Square, W.C.1, on or before December 31, 1938. Each essay must be signed with a motto and accompanied by a sealed envelope marked on the outside with the motto and containing the name and address of the author.

The prize consists of a cheque for £50 and an illuminated certificate.

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Midland Ophthalmological Society

THE officers of the Midland Ophthalmological Society for the current year are:—President, W. Niccol; Vice-President, T. E. Ashdown-Carr; Treasurer, E. B. Alabaster; Secretaries, T. Harrison Butler and L. P. Jameson Evans.

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Hunterian Lecture

Mr. J. COLE MARSHALL will deliver a Hunterian Lecture at the Royal College of Surgeons of England on January 17 at 5 p.m. The subject will be "The surgical treatment of detachment of the retina."

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The Cuban Institute for the Protection of Sight

AN institute has been founded in the city of Havana, Cuba, for the purpose of statistics on ocular diseases, and to publish papers on the most common causes producing these conditions.

The institution has been founded because the prevention of blindness and the study and publication of knowledge essential for the better preservation of vision, is above all a social problem of universal interest.

The Committee is presided over by Dr. Tomas R. Yanes, surgeon-oculist, who edits the *Revista Cubana de Oto-Neuro-Oftalmiatria*, the other members of the committee being distinguished doctors,

architects, engineers. etc., representatives of various spheres of life directly concerned with the preservation of sight.

All concerned in this foundation are to be congratulated.

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Badge Design Competition

THE Board of Registration of Medical Auxiliaries, the body responsible for the National Register of Medical Auxiliary Services, proposes to issue a distinctive badge to be worn by registered medical auxiliaries, and it has been decided to institute a competition and to offer a prize of five guineas to the person who submits the best design for such a badge. The competition is open to any person.

Entry to the competition is free and persons desiring to compete should apply, in the first instance, to the Registrar, Board of Registration of Medical Auxiliaries, British Medical Association House, Tavistock Square, London, W.C.1, when full details of the competition will be sent.

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Orthoptic Examination

AN examination in Orthoptics will be held in London on January 12 and 19. Entrance fee three guineas, payable in advance. Schools represented upon the Orthoptic Board are to send names of candidates to:—Secretary, Orthoptic Board, Royal Westminster Ophthalmic Hospital, as soon as possible.

FUTURE ARRANGEMENTS

1938

January 14.—Section of Ophthalmology, Royal Society of Medicine.

January 21 and 22.—North of England Ophthalmological Society, Prof. von Rohr's lectures at Sheffield University.

February 1.—Midland Ophthalmological Society, at Wolverhampton Eye Infirmary.

February 4.—North of England Ophthalmological Society, at Newcastle-upon-Tyne.

February 11.—Section of Ophthalmology, Royal Society of Medicine.

March 4.—North of England Ophthalmological Society, at Liverpool.

March 11.—Section of Ophthalmology, Royal Society of Medicine (Clinical Meeting).

April 1.—North of England Ophthalmological Society, at Sheffield.

April 8.—Midland Ophthalmological Society, at Nottingham Eye Infirmary.

May 27.—Midland Ophthalmological Society, at Cheltenham Eye Hospital.

June 10.—Section of Ophthalmology, Royal Society of Medicine (*Annual Meeting*).

July 7-9.—Oxford Ophthalmological Congress, at Oxford.

October 7—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital (Annual Meeting).

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *October, 1937.*

ARRUGA. Treatment of detachment of the retina.

WOODS and RANDOLPH. Treatment of ocular tuberculosis.

MUSKENS. Cortical innervation of ocular movements in the horizontal plane.

DUNNINGTON and MACNIE. Detachment of the retina; operative results in 164 cases.

STOUGH. Intra-ocular neurofibroma: report of a case.

JAMESON. Surgical management of ptosis, with special reference to use of the superior rectus muscle.

KNAPP. Formation of preretinal connective tissue in the vitreous in acute choroiditis: report of three cases.

VERHOEFF and GROSSMAN. Pathogenesis of disciform degeneration of the macula.

DASHEVSKY. Localization of changes in the eyeground and peilingation of their projections on the sclerotic.

COHEN. Bilateral metastatic carcinoma of the choroid: report of a case.

SPIEGEL and SCALA. Ocular disturbances associated with experimental lesions of the mesencephalic central grey matter, with special reference to vertical ocular movements.

American Journal of Ophthalmology. *December, 1937.*

SMELSER. A comparative study of experimental and clinical exophthalmos.

MCFARLAND, KNEHR and BERENS. The effects of anoxemia on ocular movements while reading.

LANCASTER. Lighting standards.

BORLEY aud LEEF. Removal of lead shot from the vitreous by use of the biplane fluoroscope.

BARKAN. Recent advances in the surgery of chronic glaucoma.

MONCREIFF. Some technical questions in cataract surgery.

Annales d'Oculistique. *October, 1937.*

MAGITOT and DUBOIS-POULSEN. Condemnation of the coloured index in clinical perimetry.

VOISIN. Mechanism and pathogenic value of Graefe's sign.

SÉDAN. The prolonged simulation of "petrifying conjunctivitis."

WEINSTEIN. Associated symptoms in ophthalmology.

Archiv für Ophthalmologie. Band 138.

OHM. Basis of the ocular movements.

SCHIECK. Contribution to the question of the formation of papilloedema.

PAUL. Contribution to localization ophthalmoscopy. Examination of the shape and size of the eyes and of the development of anomalies of refraction utilizing the percentage computation.

KÜHN. Measurements of the fundus.

HARMS. Local and general methods of arresting squint.

Klinische Monatsblätter für Augenheilkunde. *October, 1937.*

GASTEIGER and LIEBENAM. Contribution to dysostosis multiplex with special regard to the ocular condition.
ROHRSCHNEIDER. Delayed disease of the eyes after mustard gas poisoning.
HARTMANN. Superficial and deep (discoid) corneal inflammation after exposure to sulphuretted hydrogen in caisson workers on the North Sea coast.
SOMMER. Contribution to Krukenberg's pigment spindle and to the corneal pigment ring.
BUSACCA. Introduction of an ivory shell in the anterior chamber in keratoplasty for a large adherent leucoma.
RAAB. The question of refusion.
VELHAGEN, JUNR. The factor of convergence described by Comberg.
MACHEMER. A new skiascope band.
RUBIERA and MIRANDA. The pathogenesis of coal miners' nystagmus.
KOYANAGI. The rôle of retinal angiospasm in the development of albuminuric retinitis.
FABIAN and GEORGARIOU. Contribution to the pathogenesis of true arteriosclerotic disease of the retina.
PREVEC. A case of sarcoma of the papilla.
VON PAPOLCY. The prognosis of uveal sarcoma.

Zeitschrift für Augenheilkunde. *October, 1937.*

LINDNER. The use of a vitreous fistula.
TERTSCH. Three cases of hyaloid membrane detachment.
HOEHNE. Papilloedema from ocular hypotony.
LISCH. Participation of the eyes, especially of the occurrence of iris nodules, in neurofibromatosis (von Recklinghausen's disease).

Bollettino d'Oculistica. *October, 1937.*

MOTOLESE. Traumatic scleral rupture.
CASTELLI. Zinc peroxide. Considerations and results from its use in antitrachomatous treatment.
NOBILE. A case of papulo-erosive syphilis of the palpebral conjunctiva.
SCHUPFER. Brief reply to the work of Dr. Mecca "Some pathogenetic facts about retinitis pigmentosa."

Archivio di Ottalmologia. *July-August, 1937.*

CAMPOS. Experimental research on papilloedema.
CAVALLACCI. Granuloma of the limbus from sporotrichum.

Archivos de Oftalmologia de Buenos Aires. *August, 1937.*

MALBRÁN. Biomicroscopy of keratoconus.

Revista Cubana de Oto-Neuro-Oftalmiatria.
March-April-May-June, 1937.

ALAMILLA. Congenital corneal opacities.
AVALOS. Traumatism of the extremity of the eyebrow and its consequence on the eye.
RUBIO. Facts about ophthalmic preparations.